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A DECADE'S EXPERIENCE IN THE OPERATION OF A GROUP MEDICAL-CARE ORGANIZATION*

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THE National Health Assembly, which met last summer in Washington, D. C., reached agreement on everything except the question of national compulsory health insurance. Among the subjects agreed on was the proposal that voluntary prepayment was a sound method for financing health services.

Ten years ago such an agreement would have been impossible. At that time a storm raged within and without the medical profession over the question of prepaid health programs. The focal point of the storm was Group Health Association of Washington, D. C., a medical-care organization operating on the principles of group medical practice and the prepaid method of financing. Ten years ago programs of the type represented by Group Health Association were attacked as being forerunners of "socialized medicine." Today these programs are generally supported by all who are concerned with bringing medical care of the highest standard within the reach of all.

This article is a description and an analysis of the experience of Group Health Association from 1938 through 1947. In many respects the record is notable. Yet it is not without its shortcomings. No honest student of social welfare can afford to minimize them. It is my opinion that an honest appraisal of this experience will provide guidance to future changes in the organization of medical programs.

When the Association was organized in 1937, predictions concerning its future were of two kinds. One camp predicted that it would rapidly fold up and that its doctors would become outcast members of the medical profession. The other prophesied that this prepaid type of medical care would sweep the nation's capital because of its low-cost provisions and ensnare all the physicians in a monster octopus of regimentation and bureaucracy.

Actually, neither has happened. The Association is a respectable community organization attracting respectable residents of the Washington area to membership. Its physicians are accorded the same privileges in hospitals as other doctors in the District of Columbia and surrounding area. They have similar social status. They teach at the local universities. They are members in good standing in the District of Columbia Medical Society and the American Medical Association. Many of them have private practices, devoting only part of their time to Group Health Association member patients.

OPERATION OF MEDICAL SERVICE

The medical-service plan operates in this manner: members purchase a membership certificate and pay monthly dues. In return, with certain limitations and exceptions, members have a "blank check" on the medical services by Group Health. These services include diagnosis and treatment of ailments, major and minor surgical operations, use of facilities such as x-ray and laboratory procedures, and physical therapy. Members may avail themselves of physical checkups and other preventive care. Child care and obstetric service are provided. Eye examinations are given. Members may purchase lenses and frames through the Association at a reduced cost, and they may obtain prescriptions, drugs, vitamins and other pharmaceutical supplies at the pharmacies at a saving.

There are two types of membership: full service and nonhospitalization. Those who have full service pay larger monthly dues than those who have nonhospitalization memberships. Full-service members are eligible to receive hospitalization when necessary for sixty days in any calendar year, or in any one illness or condition, or continuous period of hospitalization. This includes a semiprivate room, general nursing, use of the operating or delivery room, services of the anesthetist, dressings and most medications, routine laboratory examinations, nursery care, emergency-room facilities and the use of an ambulance as requested by the attending physician.

*Nothing in this article is to be construed as an official pronouncement of the Bureau of the Census.

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A nonhospitalization member is entitled to all the services except the hospital services mentioned above.

Changes in Benefits

Over the course of the Association's history, two divergent trends in the scope of benefits under its service program have appeared. One is toward more and more restrictions and limitations, and the other is toward greater liberalization of its program.

Restrictive trend. When the first set of by-laws was adopted on March 22, 1937, there were relatively few restrictions on service. Only two important restrictions were embodied in the first set of by-laws: if a member became ill outside the Washington area, the Association reimbursed the member for expenses only up to the amount that would have been incurred had the member resided in the Washington area; and if a member's sickness was caused by a third person and the third person was required by law to pay for the member's sickness, the Association was entitled to reimbursement for the medical service furnished to the member.

As the Association developed, a gradual but ever-growing set of restrictions was adopted. Table 1 presents a partial list showing the development of these restrictions.

Liberalization trend. On the other hand, there have been many examples of a liberalization in the service program. The first set of by-laws restricted membership to Government employees. In 1946 other employees became eligible to join on a group basis, and in 1947 they became eligible to join on an individual basis.

The first set of by-laws restricted house calls within a radius of ten miles of the center of the District of Columbia. At the present time the radius is fifteen miles.

Other examples of liberalization of the service program are: night clinics (tried for a time but discontinued); delivery service of pharmacy supplies; night telephone service; discount of 5 per cent for a year's prepayment of dues and 2 per cent for six months' prepayment; routine physical examinations encouraged; service to refugee children without cost; early infant-care classes and prenatal classes (not in operation at the present time); members drafted into military service could suspend membership; racial barriers dropped, and Negroes accepted for membership; health education forums (continued for a time but not in operation at the present time except for a series of psychiatric counseling forums on a fee basis); availability of optical service for the purchase of eyeglasses and frames at reduced cost; waiting period for service removed; and availability of child-guidance service.

TABLE 1. *Partial List, Showing Development of Restrictions on Services Provided by Group Health Association.*

EFFECTIVE DATE	RESTRICTION	SOURCE
1937	Medical service not to include: treatment of industrial accidents; operations on the brain or nervous system; mental cases, tuberculosis, drug or alcohol addiction.	*Minutes*, Board of Trustees, April 6, 1937
	Members to pay for the following items: medicines, drugs or surgical appliances; radium and deep-x-ray treatments; oxygen tanks, tents or materials; blood transfusions.	
1938	$1.00 charge for first house call in any illness	*GHA News Letter*, September, 1938
	$25.00 charge in maternity cases	
June 1, 1939	Members to pay $1.50 for x-ray film and material	*Minutes*, Board of Trustees, May 24, 1939
August 1, 1939	Members with ailments or physical conditions requiring treatment, present at date of admission, to reimburse Association at cost for service rendered in connection with treatment	*Minutes*, Board of Trustees, July 24, 1939
1939	During first 10 months of service, members to reimburse Association for cost of services, including hospitalization, connected with ailments present at admission, confinement and elective operations	*Minutes of Special Membership Meeting*, November 21, 1939
September 3, 1940	Additional services not covered: plastic surgery, correction of deformities and birthmarks, psychiatric treatment and chiropody.	*Minutes*, Board of Trustees, September 9, 1940
August 1, 1941	Charge for obstetric care increased to $50.00*	*Minutes*, Board of Trustees, June 23, 1941
January 1, 1948	Those who have all services at cost except clinic consultations and routine laboratory services and all those 60 years of age or over admitted after January 1, 1948, are now restricted for all services, — no exceptions, — dues to apply against cost†	Personal communication from Hospitalization Secretary, March 3, 1948

*At the present time this additional charge for obstetric care is $100.00.

†Interestingly enough, the suggestion was made as early as 1939 that elderly dependents be required to pay for all services at cost. (*Minutes*, Board of Trustees, September 18, 1939).

MEMBERSHIP ELIGIBILITY

At the present time membership is open to all residents of the District of Columbia and vicinity. There are no restrictions concerning sex, race, occupation or place of employment. Up until January 1, 1948, there were no age restrictions, but on that date the restriction was imposed as noted in Table 1.

Participants may join on an individual basis or as part of a group.

Individual Enrollment

Individual applicants pay a $2.00 application fee. This fee covers the expense involved in processing the application and in making laboratory tests such as urinalysis and blood tests.

Group Enrollment

No application fees are paid in a group enrollment. A group consists of employees of a single employer, or persons in one administrative unit of a business or Government agency or persons in one building or office. The percentage of those who must join to obtain group benefits varies according to the size of the basic unit.

Whether a person joins on an individual basis or with a group, a $10 fee must be paid. This is the membership-certificate fee and gives the member voting and participating privileges in the organization. The $10 membership fee covers all persons in a family unit unless more than one person desires to take out a membership certificate. The fee, which is the member's contribution toward the cost of the facilities and equipment purchased by the Association, is not refundable, but members who leave the Association may transfer their membership certificate under certain circumstances.

Through the membership certificate members have an equal share in ownership, and through annual elections of the board of trustees and votes on specific issues, as submitted to them, they determine the type and general character of its activities.

TABLE 2. *Membership Growth, Group Health Association, 1937–1947.*

YEAR	FIRST OF MONTH	NO. OF MEMBERS	NO. OF DEPENDENTS	TOTALS PARTICIPANTS
1937	Nov.	900	*	*
1938	Nov.	2,355	3,035	5,390
1939	Jan.	2,115	2,759	4,874
1940	Jan.	2,383	3,166	5,549
1941	Jan.	2,791	3,819	6,610
1942	Jan.	3,284	4,509	7,793
1943	Jan.	3,375	4,957	8,332
1944	Jan.	3,566	5,570	9,136
1945	Jan.	3,253	5,427	8,680
1946	Jan.	3,362	5,765	9,127
1947	Jan.	4,097	7,457	11,554
1947	July	4,853	7,826	12,679

*Figures not available.

GROWTH IN MEMBERSHIP AND MEMBERSHIP TURNOVER

Table 2 shows the gradual rise in membership during the first ten years of operation. During the early years, the growth was fairly steady. During World War II, members were permitted to add dependents but new applications for membership were not accepted because of the difficulty in obtaining additional physicians and auxiliary medical personnel for the staff. When World War II ended, membership rolls were again opened and nongovernmental employees were permitted to join on a group basis. In February, 1947, any resident of the District of Columbia and vicinity was permitted to join on either a group or an individual basis.

By expansion of the promotion department and through an aggressive membership drive, large gains in membership were recorded in 1947. The most notable of these gains was due to the signing of contracts between Group Health Association and the Food and Agriculture Organization, the International Bank for Reconstruction and Development and the International Monetary Fund. Under these agreements, the Association enrolled the employees of these agencies as group members.

TABLE 3. *Rates of Accession and Secession in Membership, August, 1946, Through September, 1947.*

DATE	NO. OF PARTICIPANTS AS OF END OF MONTH	NO. OF ADMISSIONS	RATE OF ACCESSION	NO. OF WITHDRAWALS	RATE OF SECESSION
			per 100 participants		*per 100 participants*
1946:					
August	10,298	451	4.38	140	1.36
September	10,500	309	2.94	107	1.02
October	10,761	382	3.55	121	1.12
November	10,970	369	3.36	160	1.46
December	11,188	328	2.93	110	0.98
1947:					
January	11,666*	270	2.31	158	1.42
February	12,073	474	3.93	67	0.55
March	12,216	213	1.74	70	0.62
April	12,177	235	1.93	274†	2.25
May	12,206	463	3.79	434†	3.56
June	12,546	594	4.73	254	2.02
July	12,679	435	3.35	302	2.38
August	12,806	360	2.81	233	1.82
September	13,522	974	7.20	258	1.91
Fourteen-month average			3.47		1.63

*This figure was adjusted by actual count of membership, as a result of which 266 additional participants were "picked up" on the membership rolls.

†Withdrawals are concentrated owing to extensive cleaning out of pending withdrawals that had been hanging fire for six months or more.

Increase in membership through admission of new applicants has been retarded by the withdrawal of members who resign or suspend the membership of a dependent for one reason or another. For instance, during the fourteen months from August, 1946, to September, 1947, for every 7 new participants added, about 3 old participants withdrew. (The actual figures are 3.47 new participants as against 1.63 withdrawn participants and are derived from Table 3.) According to Miss Dorothy Sharrow, administrative assistant to the executive director, about 90 per cent of those who withdraw give as reasons the fact that they are moving away from the city. However, the extent to which other factors have caused members to withdraw or suspend membership is analyzed below.

MEMBERSHIP CHARACTERISTICS

Family Status

By types of family unit, the largest proportion of memberships consisted of member, spouse and one or more children (Table 4).

A comprehensive survey made in 1942 showed that at that time approximately a third of the participants were in the "member only" category, another third comprising the "member and spouse only" group.[2]

In 1947 the average number of participants in each membership was 2.6.[1] In 1942 it had been 2.3.[2]

Though the average size of family unit has increased from 1942 to 1947, the families* are small, compared to those in an urban population.

Other important features of the 1942 survey showed that participants, compared to an urban population group, consist of a larger proportion who are married and a smaller proportion widowed or divorced.[2]

TABLE 4. *Distribution by Types of Family Unit, March, 1947.**

TYPE OF MEMBERSHIP	PERCENTAGE
Member, spouse and children	45.7
Member only	27.1
Member and spouse only	19.5
Other (all types of family units, including adult dependents)	7.7

*Based on *Number and Composition of Group Health Membership.*[3]

Age of Participant

A sample study of the ages of members was made in 1947 by examination of the entrance age of every fifth member participant who joined between May, 1944, and July, 1946. The ages at time of entrance are presented in Table 5, which shows that over 90 per cent of new member participants were under 45 years of age at the time of the study.

TABLE 5. *Entrance Age of Sample Group.*

AGE	NO. OF MEMBERS	PERCENTAGE OF TOTAL
yr.		
Newborn infants	27	6
0–4 years	66	14
5–20 years	59	12
21–44 years	289	61
45–59 years	29	6
60 and over	4	1
Total	474	

The more comprehensive study made in 1942 showed, by actual count, that half the members were between 20 and 34 years of age and only an eighth 50 years old or over. The median age for all participants was 29.7 years, the median age for male participants being 30.1 and that for female participants 29.3.[2]

*All members of a family are not always included as a member's dependent for service, but it is my impression that the number of dependents who are not listed is very small.

DUES AND FEES

The cost of monthly dues depends on the type of membership and the size of the member's family. The present rates are shown in Table 6.

Limitations and Exceptions Under Dues System

Obstetric care. When obtaining prenatal, delivery and post-partum care, the member pays the first $100 toward the hospital bill when both parents have been listed for membership for ten months prior to expected delivery. When obstetric service is obtained in cases in which delivery is expected before ten months of continuous membership, the member meets the entire hospital bill and pays to the Association an additional $75.00.

Cost of Materials

The expenses of materials, such as x-ray films, serums, eyeglasses and lenses, are paid by members. These are usually purchased from the Association's pharmacies or Optical Department. There are special charges for electrocardiograph materials, basal-metabolic-test materials, x-ray therapy, physical therapy and laboratory test materials. Some of these material charges appear to be actually service charges — for example, x-ray and physical therapy. A charge of $1.00 is placed on each x-ray and physical-therapy treatment rendered, "to compensate for heavy wear on expensive equipment," according to the *Minutes* of the Board of Trustees (July 8, 1947). If these *are actually* service charges the principle of removing barriers to medical care is being violated because service charges are apt to constitute "hesitation fees" — that is, fees that cause participants to neglect medical attention because of additional costs involved.

TABLE 6. *Cost of Dues.*

PARTICIPANT	MONTHLY DUES	
	WITH HOSPITALIZATION	WITHOUT HOSPITALIZATION
Member	$3.50	$3.00
Adult dependent	3.50	3.00
Child dependent (for each of first three children — no charge for more than three children)	2.25	2.00

Restricted services. On the basis of laboratory tests, medical histories and physical examinations, certain types of medical conditions existing prior to membership are determined. Members who join on an individual basis (nongroup) may be restricted for services under the monthly dues for specific conditions present at time of admission. Service for these restricted conditions is given on a cost basis.

Limitations of Service

There is some evidence that the present rates of dues and other fees are so high that the Associa-

tion is attracting only people with the highest family incomes.

In 1939 the median salary of members was $2700, and 53 per cent of the members had salaries under $3000 per year. A survey for 1946 indicated that the median family income was about $5600.

The original purpose of GHA was to provide the best type of medical care to those who usually could not afford it. What is needed is a comprehensive study to determine whether the program has become "selective" in its operation.

Quality of Medical Service

In his report at the annual membership meeting in February, 1947, former president Harry J. Becker stated:

> We have nearly completed the recruitment for the medical care organization which a year ago was largely a dream on paper. We are happy to report that we have been able to recruit professional staff to meet substantially the medical care requirements for our membership. For the first time in a long while we have as many general physicians as are necessary to maintain our medical standards. Medical judgment will support my statement that the members of Group Health are today enjoying a better quality of medical care than can be purchased on either a prepaid or fee-for-service basis elsewhere in Washington. We have available to any member of the Washington community the most completely equipped and staffed out-patient service in Washington utilizing the principle of the group practice of medicine.

It is beyond the scope of this article to pass medical judgment on the caliber of the Association's medical staff. However, one method of appraising the quality of the medical care program is by considering member reaction to the program.

A survey made in the summer of 1947 indicated that the three aspects of the program most appreciated by the members were the competence of the medical staff; the prepayment features of paying for medical care, which afforded insurance against large medical bills; and the scope of service available.

However, member reaction to the program has often been negative. A study of the records of separation for four selected months (Table 7) indicated that from 4 to 20 per cent of those who are separated in any one month are separated because of dissatisfaction with the service. It is also possible that there are among those who give no reason for requesting separation members who are dissatisfied with the service but who prefer not to state their reasons. (On the other hand, the administrative assistant to the executive director has stated that 90 per cent of current separations are due to the fact that participants are leaving the Washington metropolitan area.)

Unsatisfactory service does not always lead to the stage where the member requests separation. For the ten months from June, 1946, through March, 1947, the Complaints and Suggestions Committee received 54 complaint letters and 16 letters suggesting ways of improving the service.

Complaints and suggestions are usually received by the Association in writing to assure accurate transmittal of the members' view.* They are routed to the executive director, who consults interested parties, gets other information as needed and takes such action as he considers to the best interests of the Association. The Complaints and Suggestions Committee at its monthly meetings reviews the papers, searching for factors that may have been overlooked. The purpose is to determine the essential nature of the complaint and find underlying causes.

Table 7. *Separations by Suspension or Resignation for Selected Months.*

Reason	No. of Participants	Percentage of Total Separations
April, 1946:		
Leaving area	40	30
Receiving medical care elsewhere	10	9
Dues increase	4	3
Dissatisfied with service	21	20
Not given	31	29
Total	106	
December, 1947:		
Leaving area	72	46
Receiving medical care elsewhere	1	1
Dissatisfied with service	6	4
Not given	18	11
Other (deceased, membership transferred to dependent and so forth)	61	38
Total	158	
January, 1948:		
Leaving area	100	63
Receiving medical care elsewhere	5	3
Dissatisfied with service	6	4
Not given	31	19
Other	18	11
Total	160	
February, 1948:		
Leaving area	64	43
Receiving medical care elsewhere	6	4
Dissatisfied with service	6	4
Not given	60	41
Other	11	8
Total	147	

Many items of criticism and suggestions for improving the service were received in a recent survey. Most of all, members wanted the Association to expand its services to include dental care, a psychiatric program and other medical facilities not now provided on a prepaid basis. The two greatest limitations on the program, according to member opinion, are its telephone service and its appointment scheduling.

A survey made in 1942[2] revealed the following:

> The turnover of membership which occurred prior to 1942 affected primarily the younger group. The older members showed a greater degree of stability. . . . Almost two thirds [of the participants] who were 50 years old or over on January 1, 1942, joined the organization before

*Obviously, not all complaints are reduced to writing, and many complaints never reach the organization.

January 1, 1939, whereas only 38% of the present membership as a whole joined before that date. Less than 5% of the members who joined in 1941 were 50 years old or over, but 20% of the present membership who joined before 1939 are in that age group now.

Infant and Maternal Mortality and General Death Rates

Infant Death Rate

A reliable barometer of the health of any segment of the population is the infant mortality rate of the group. This index is the number of infant deaths per thousand live births. An infant death is a death occurring when the child is less than one year of age and does not include stillbirths.

According to Sir Arthur Newsholme, an outstanding British authority on public health, "Infant mortality is the most sensitive index we possess of social welfare. If babies were well-born and well-cared for, their mortality would be negligible."

Dr. George C. Ruhland, health officer for the District of Columbia, terms infant mortality "the most delicate indicator of the health mindedness of a community and the nation."

Data on infant mortality for babies born to members are available and are reproduced in Table 8, compared to the infant mortality rates for the white population in the District of Columbia.

Since the composition of the Association is almost entirely white, it was believed that a more valid comparison would result by the use of infant mortality figures for the white population in the District of Columbia rather than the general (Negro and white) average.*

*It is generally agreed that there is a great disparity in the relative health level of Negroes and whites. The life expectancy of Negroes is approximately ten to twelve years shorter than that of whites; approximately three times as many Negro males and four times as many females, proportionately, die of tuberculosis. The death rate from heart disease is also considerably greater among Negroes than among whites.

Table 8. *Infant Mortality, Group Health Association and White Population of the District of Columbia, 1937-1946.**

Year	Group Health Association			District of Columbia White Population
	No. of live births	No. of infant deaths	Rate per thousand live births	Rate per thousand live births
1937†	—	—	—	—
1938†	—	—	—	—
1939	109	1	9.1	33.6
1940	151	4	26.0	36.8
1941	233	4	17.0	38.7
1942	281	6	21.0	39.2
1943	309	3	8.2	28.5
1944	323	6	18.6	27.7
1945	256	0	0	29.6
1946	345	5	14.0	29.5
Totals	2,007	29		
Averages			14.2‡	32.9

*Figures for Group Health Association supplied by Statistical Department, Group Health Association, and figures for the District of Columbia by Health Department, District of Columbia.

†No data available for Group Health Association.

‡The standard error of this arithmetic mean is 2.734. "Knowledge of this standard deviation, or standard error," says Mills,[3] "enables us to set limits within which it is highly likely that the true mean lies."

From Table 8 it will be seen that for every year for which data have been recorded (only data missing are those from November 1, 1937, the date of initial operation of the Group Health Association, to December 31, 1938), the Association's infant mortality rate is lower than that for the white population of the District of Columbia.

Taken by themselves, the infant mortality rates appear remarkably low. From 1939 to 1946, the average was 14.2 deaths per thousand live births per year (standard error of deviation 2.734) as compared with an average of 32.9 infant deaths per thousand live births for the white population of the District of Columbia during the same period. It would be still more remarkable, however, if the results were less striking in view of the fact that the family incomes of members are probably higher than those of the "average" white family in the District of Columbia.

Adequate incomes make possible healthful living, provide proper diets for expectant mothers and permit needed rest for pregnant women. The relation between economic factors and health has often been stated but rarely more effectively than in a recent radio broadcast:

> . . . What is health? It's not the great operation. It's not the surgeon and his sterile rubber gloves. It's not a test tube full of vitamins. Vitamins, yes. But not in test tubes . . . in people. Health is day to day living; the food you eat, the clothes you wear, the house you live in. The size of the town in which you're born. The kind of water supply and sewage disposal. Is the road to your house good enough so the doctor can get there on a rainy day? Health is the number of dollars a week your father earns. Health is the teacher in the fourth grade who sees you squinting and tells your mother. Health is love and understanding when you're an infant. All these things. Many more. You can't solve it with a quick dramatic operation. . . .[4]

Table 9. *Maternal Deaths for White Population of District of Columbia, 1937–1946.*

Year	Rate per Thousand Live Births
1937	3.6
1938	4.2
1939	3.7
1940	2.4
1941	2.3
1942	1.7
1943	1.3
1944	0.8
1945	0.5
1946	0.5

I do not wish to belittle the competence of the Association's obstetric staff or the adequacy of the prenatal, delivery and postnatal care given to members. However, since members use the same hospital facilities as other white persons in the community, and since Group Health Association obstetricians and pediatricians are not supermen, the remarkable difference between infant mortality rates shown in Table 8 can be partially ascribed to

the suspected high family income levels of members mentioned above and the fact that, with very few exceptions, births occurred in the hospitals rather than in homes.

This last factor was a greater influence in explaining the difference in infant mortality rates in the early years of operation from those in recent years. The proportion of hospital deliveries of white mothers in the District of Columbia has been steadily increasing during the past decade. In 1937, 95.1 per cent of births for the white population occurred in hospitals; in 1946, 99 per cent of these births occurred in hospitals.

Maternal Death Rates

The maternal death rate is another index of general health effectiveness in a community.

About 2000 babies have been born to Group Health Association mothers in the last ten years without a maternal death. For the white population in the District of Columbia, maternal death rates per thousand live births are shown in Table 9.

General Mortality Rates

Table 10 shows the general mortality rates for Group Health Association members from 1938 to 1946. For purposes of comparison the mortality rates for the District of Columbia for all causes, all colors, and those for all causes in the white population are listed in Table 11. It will be seen that the death rates among Association participants are lower than those for the District of Columbia for every year for which statistics of Group Health Association mortality are available.

TABLE 10. *Mortality for Group Health Association Members, 1938–1946.*

YEAR	TOTAL NO. OF PARTICIPANTS AS OF JANUARY 1	AVERAGE NO. OF PARTICIPANTS FOR YEAR*	NO. OF DEATHS	RATE PER THOUSAND PARTICIPANTS
1938	?	—	5(?)	—
1939	4,874	5,211	24	4.61
1940	5,549	6,079	15	2.47
1941	6,610	7,210	17	2.36
1942	7,793	8,062	22	2.73
1943	8,332	8,734	22	2.52
1944	9,136	8,908	30	3.37
1945	8,680	8,903	19	2.14
1946	9,127	10,046	23	2.29
1939-1946	—	63,150	172†	2.72

*Computed by use of two-year moving averages with exception of figure for year 1946. In 1946, the number of members is an average of the membership levels at the end of each month of that year.

†Five (?) deaths in 1938 omitted from total.

As shown in Table 10, the average mortality rate for the years 1939–1946 for Group Health Association members is 2.72 per thousand population. However, when specific death rates* are applied to the composition of the membership for 1942 (the only year for which the composition of membership by sex and age is available), it is found that the expected death rate is 6.57.

In other words, part of the low death rate is explained by the fact that, on the basis of the composition of its participants as of January 1, 1942, its members are relatively young, women preponderate, and the participants are predominantly white.

However, in view of the extraordinarily low death rate over a period of years, it may be concluded that factors other than age, sex and race are responsible. What these factors are and to what extent they influenced the mortality rate are not definitely known. It can only be surmised that these factors include the relatively high socioeconomic levels of participating families and the arrangement under which members obtain their medical care.

TABLE 11. *Mortality Rates of Group Health Association Members Compared with Those for Others in the District of Columbia, 1939–1946.*

YEAR	GROUP HEALTH ASSOCIATION RATE	DISTRICT OF COLUMBIA RATE*	
		ALL CAUSES ALL RACES	ALL CAUSES WHITE RACE
1939	4.6	13.0	11.2
1940	2.5	13.3	12.1
1941	2.3	11.8	10.5
1942	2.7	11.1	9.4
1943	2.5	10.9	9.8
1944	3.4	10.0	8.6
1945	2.1	10.4	8.7

*Source: Department of Health, Washington, D. C.

*A specific death rate is one for a selected population group, usually based on race, sex and age.

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COARCTATION OF THE AORTA ASSOCIATED WITH ADAMS–STOKES SYNDROME, COMPLETE HEART BLOCK AND BICUSPID CALCAREOUS AORTIC VALVE

Report of a Case

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TO OUR knowledge coarctation of the aorta in association with complete heart block and Adams–Stokes syndrome has not previously been reported. The patient in the case reported below was first observed while having attacks of Adams–Stokes syndrome. Examination revealed signs characteristic of coarctation of the aorta. Varying degrees of heart block were manifest and subsequently became complete. Post-mortem examination showed, in addition to coarctation, a calcified bicuspid aortic valve. The cause of the heart block prompted considerable debate prior to death.

REVIEW OF THE LITERATURE

Coarctation of the aorta has been very thoroughly discussed in its clinical and pathological aspects. Virtually all autopsied cases in the literature were assembled by Abbott[1] up to 1928, and subsequent cases over two years of age up to 1946 by Reifenstein, Levine and Gross.[2] From these reviews it may be observed that bicuspid aortic valves are of such frequent occurrence as to be almost considered a part of the coarctation syndrome; in the first report they were found in 25 per cent of 183 cases[1]; in the second this anomaly was present in 42 per cent of 104 cases.[2] It is emphasized that these bicuspid valves are a vulnerable focus for subacute bacterial endocarditis and other types of valvulitis. Among the patients living to adult age, the development of a calcareous aortic valve is not unusual; Reifenstein et al.[2] described 11 cases of "calcific aortic stenosis" among 93 patients, in which fairly complete valvular descriptions were available.

The subject of complete heart block in younger age groups has recently been discussed by Crawford and Di Gregorio,[3] who itemize various possible causes and review the literature of complete heart block due to congenital heart disease. The following criteria have been set up for congenital heart block: proof by graphic methods; knowledge of a slow pulse at an early age; signs of congenital heart disease, and absence of a history of infection or other factors that might produce heart block. Fifty cases have been reported in which complete heart block has appeared to be of congenital origin. Yater et al.[4, 5] have reviewed and reported the greater number of these. The associated congenital lesions have included ventricular septal defects as the most common (26 out of 44 cases),[4] patent ductus arteriosus, auricular septal defects, pulmonary stenosis, transposition of the great vessels, common aorta and pulmonary artery, and cor biatrium triloculare.

Waldman[6] reported a case of patent ductus arteriosus with transient heart block and mild Adams–Stokes attacks. This was the first such case of congenital heart disease in which a cardiogram was obtained during an attack. Faessler[7] reported 8 cases of Adams–Stokes syndrome with congenital heart disease, 6 patients having complete block between attacks; of these, 6 cases were diagnosed as ventricular septal defects, 1 as a patent ductus, and 1 as a persistent foramen ovale. Waldman[6] points out that heart block in congenital heart disease may be present as a part of the congenital defect or may be acquired and superimposed upon the congenital deformity. Prior to post-mortem examination it might have been argued that the case reported below could fit into the former category, but subsequently the latter explanation appeared probable.

Complete heart block in calcareous aortic stenosis has recently been reviewed by Warshawsky and Abramson.[8] They report a case in a fifty-two-year-old patient with rheumatic heart disease manifested by aortic stenosis and insufficiency, showing calcific extension from the aortic valve into the membranous portion of the interventricular septum. Dry and Willius,[9] in a study of calcareous aortic stenosis, found 1 case, out of 63 examined post mortem, that showed complete heart block with a nodular excrescence of calcium extending from the mitral cusp of the aortic valve in such a manner as to involve the bundle of His. In their series of 85 cases with marked clinical evidence of calcareous aortic stenosis, 2 showed complete heart block, and 5 others, delayed auriculoventricular conduction. In a study of 22 cases of calcific aortic stenosis, Reich[10] found 1 case of rheumatic etiology that showed complete heart block and left-bundle-branch block. Boas[11] reported 2 out of 6 cases of aortic stenosis that showed complete heart block. Cohen et al.,[12] in an analysis of 9 cases of calcareous aortic stenosis, described 1 with complete heart block.

CASE REPORT

L. A. M., a 31-year-old married schoolteacher, was first seen by one of us (R. J. C.) in August, 1946. He was considered to have been normal at the time of birth. He had had a vague illness at the age of 10 for about 10 days, ano-

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†Pathologist, National Institute of Health, United States Public Health Service; formerly, resident pathologist, Massachusetts General Hospital.

ciated with leg pains; a heart murmur was noted, and his pulse was said to have been unusually slow, but no exact details were obtainable. Through school and college he led a very active life. He was an ardent tennis player and participated in all types of sports.

In 1939 the patient was rejected for the Navy because of an elevated blood pressure. Consequently he was examined at the Massachusetts General Hospital Out-Patient Department (U. No. 223487). Examination at that time showed a regular cardiac rate at 70, systolic and diastolic murmurs at the base and apex of the heart, and a blood pressure of 180/65 in both arms whereas in the legs the systolic pressure was 140. Diagnosis was deferred, and the patient was sent for chest x-ray films, which were reported as showing hypertrophy of the left ventricle without evident rib changes (November 15, 1939). The patient never returned for follow-up examination.

In 1943, when he again attempted to enlist in the Navy, he was told that his blood pressure was 230/80 and that he had coarctation of the aorta. He remained entirely asymptomatic and led an active life until May, 1946. At that time, while working under considerable pressure, he first experienced a blackout, during which he had a mild generalized convulsion and became unconscious for about a minute. This type of episode was repeated on frequent occasions in subsequent weeks. An electroencephalogram taken on suspicion of epilepsy was stated to have been normal.

In July an electrocardiogram was reported as showing complete heart block, and it was believed that the seizures were of the Adams–Stokes type. Treatment with ephedrine and atropine was followed by a temporary decrease in the number of attacks.

In August, because of blackouts increasing in frequency up to several an hour, as well as "brownouts," consisting of weakness and faintness without convulsive movements or loss of consciousness, the patient was referred to us.* He denied joint pains, symptoms of infection, dyspnea, chest pain or any other symptomatology.

The family history indicated that his father and mother were both living and well; two siblings were in excellent health. He had been married for 5 years and had a 2-year-old child; his wife and child were well.

Physical examination revealed a thin but fairly well developed man appearing tense and concerned over his condition but not acutely ill. The neck vessels showed a vigorous collapsing type of arterial pulsations but no abnormal venous fullness. The cardiac apical beat was forceful and felt in the fifth interspace 1 cm. outside the midclavicular line. There was a harsh Grade II to III systolic murmur at the base, heard loudest in the second left interspace, but radiating all over the precordium, especially toward the lower sternal areas. The murmur radiated into the neck vessels and was heard loudly over t e upper dorsal spine. Also at the base there was a high-pitched, Grade II, blowing diastolic murmur heard equally to the right and left of the sternum in the second interspaces and radiating down the sternal borders. The aortic second sound was greater than the pulmonic second sound. The first sound at the apex was forceful but variable in intensity, and a Grade II systolic murmur was heard in this area, together with a rather loud third sound. No thrill was made out. Definite intercostal pulsations were felt in the posterior axillary line. The lungs were clear and resonant. The abdomen was not remarkable; the liver and spleen were not felt. The femoral and dorsalis pedis pulsations were palpable but definitely reduced.

The pulse rate at the time of examination was 48, and somewhat irregular. The temperature was normal. The blood pressure in both arms was 200/60. The systolic blood pressure in the legs was 100, and the diastolic was questionable at 70.

During the examination the patient had no blackouts. An electrocardiogram made at the time showed varying degrees of heart block ranging from complete dissociation to simple first-degree block with a varying PR prolongation. The basic pattern was the same as that shown in Figure 1.

The patient was admitted to the Winchester Hospital for further observation and treatment. Routine blood and urine studies were not remarkable. A blood Hinton test was negative. Three blood cultures were negative. Electrocardiograms on various occasions showed normal sinus rhythm at a rate of 90 with a PR interval of 0.22 second as demonstrated in Figure 1 *A*; and again, complete auriculoventricular dissociation with a ventricular rate of 45, as in Figure 1 *B*. Unfortunately, no tracing was obtained during an attack. X-ray studies of the heart showed prominence of the left ventricle, with unusually vigorous contractions. The aortic knob was small. The left auricle was not remarkable. The transverse diameter of the heart measured 13.9 cm., as compared with an internal chest diameter of 28.2 cm. The lung fields were normal. There was slight but

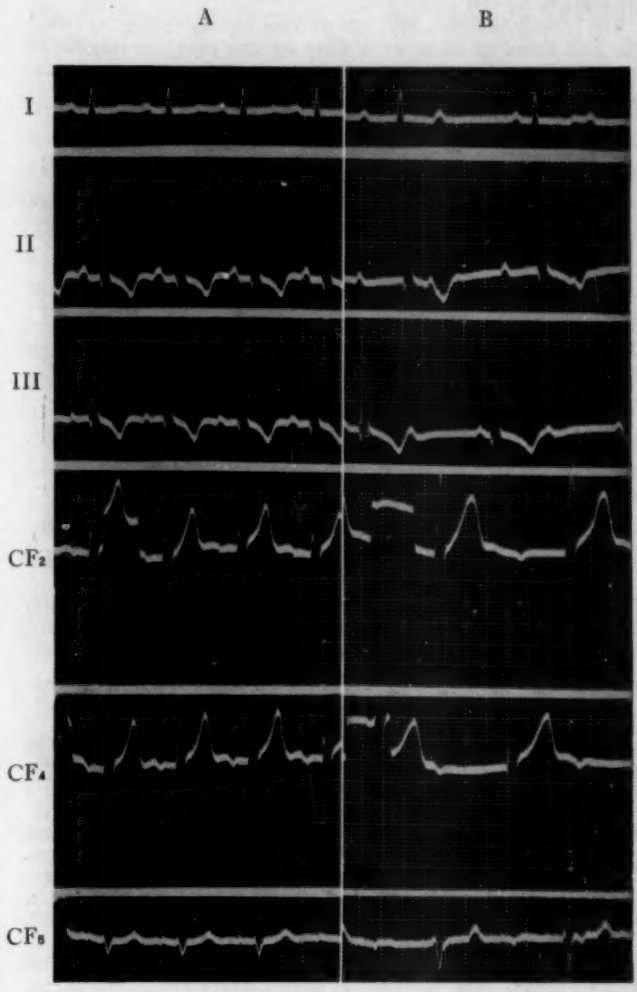

FIGURE 1. *Electrocardiograms in a Case of Coarctation of the Aorta.*

A shows regular sinus rhythm at a rate of 90, with a PR interval of 0.22 second; the T waves are low and upright in Lead 1, inverted in Leads 2 and 3, with sagging ST segments and 1.5-mm. Q waves and prominent T waves in Leads CF2 and CF4, with relatively low R and deep S waves. The R wave is very low in Lead CF5, with upright T waves. B shows complete auriculoventricular dissociation, with a ventricular rate of 45 and an auricular rate of 90.

definite notching of the ribs. The temperature remained normal. The blood pressure in the arms varied from 190/50 to 210/60. Two milligrams of epinephrine in oil was given intramuscularly twice daily, and 0.65 mg. of atropine by mouth thrice daily. There was continued shifting in the heart rate from about 90 to about 40, several times daily. At intervals the patient had mild "brownouts," but none of the blackouts.

After a week of hospitalization he was discharged on ephedrine sulfate, 48 mg. three times daily, and atropine sulfate, 0.65 mg. four times daily by mouth. The first week at home he had two mild blackouts, but subsequent to this he had

*We are indebted to Dr. J. M. Wilcox, of Woburn, Massachusetts, who referred the patient and kindly consented to his being followed thereafter.

only "brownouts" occurring with shifts in pulse rate from 80 to 40, which occurred off and on once or twice daily. In September he resumed teaching. In late October his heart was remaining in complete block most of the time. By mid-November his rhythm settled into permanent block at a rate of 40, and he was quite symptom free. During the winter, spring and summer he led an active life, often walking 5 miles a day. His blood pressure in the arms gradually increased up to 290/50. The heart size increased 4 cm. in the transverse diameter. Complete heart block with a ventricular rate of 40 persisted. Apart from some increase in nervous irritability he felt well.

On November 16, 1947, the patient got up feeling as usual. He had flown to New York City on the previous day to attend a football game. In midmorning he remarked that

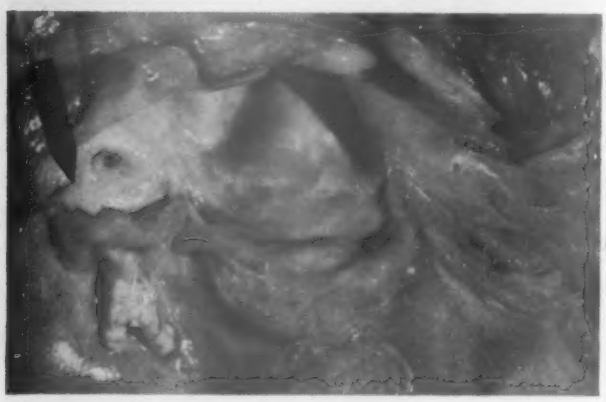

FIGURE 2. *Gross Specimen of the Aortic Valve.*

The arrow indicates the rudimentary ridge between two coronary cusps; the right coronary cusp is on the left, and most of the left is on the right of the plate. (Note A-shaped calcific mass below.)

his heart felt as though it were "going to be jumpy," and he took a dose of atropine. He seemed nervously irritable. On returning from church he sat down to read the paper. He suddenly put the paper down and stated that he felt smothered; his eyes rolled upward, and he took several short gasps, became pallid and apparently expired within 2 or 3 minutes.

Post-mortem examination revealed a well developed man, 5 feet, 7 inches, in height and weighing about 150 pounds. He appeared at least 5 years younger than his actual age of 32. Slight edema was evident in the lower legs and ankles.

Examination of the thoracic organs showed light and doughy lungs. The pericardial cavity contained about 20 cc. of clear, yellow fluid. There was a fibrous adhesion at the apex of the heart between the visceral and the parietal pericardium occupying an area 1.5 cm. in diameter. The heart was enlarged and weighed 630 gm. The left ventricle was markedly elongated; the distance from the mitral ring to the apex of the left ventricle was 10.2 cm., compared to 6.7 cm. for the distance from the tricuspid ring to the apex of the right ventricle. The wall of the left ventricle was thickened to 21 mm. at the mitral valve, but it tapered to only 4 mm. in thickness at the apex. The right ventricle appeared slightly dilated, and the wall measured 4 mm. in thickness. The foramen ovale was closed, and there were no interventricular or interauricular defects.

The aortic ring measured 7 cm. in diameter. The valve was bicuspid, sclerotic and stenotic, the cusps being stiff and thickened, with rolled edges. There was considerable calcification, especially at the bases of the common coronary cusps and extending into the aortic wall in the sinuses of Valsalva. In addition, a rough, yellow, inverted Y-shaped, hard, verrucous, calcific mass extended down over the left ventricular endocardium from the right coronary cusp. This mass was superficial and did not impinge on the underlying myocardium.

The two coronary cusps were equally small and confluent, with a rudimentary ridge instead of a commissure between them (Fig. 2). Together these cusps measured only slightly more in transverse diameter than the opposing noncoronary cusp. The rudimentary septum partially dividing the small coronary cusps was smooth and hemispherical and projected 2 mm. above the sinus wall. This ridge ran vertically and joined the aorta much lower than the other two commissures, which showed no interadherence between the cusps. In the anterior leaflet of the mitral valve just below the noncoronary cusp there was a rough, atheromatous plaque, which measured 1.5 cm. in diameter. The remainder of the mitral valve, together with the pulmonary and tricuspid valves, was entirely normal.

The stiffness of the "double" aortic cusp and the absence of the normal commissure left the coronary orifices completely unprotected; however, the coronary arteries contained only minimal atherosclerotic changes. The right coronary artery had two ostia. The ascending aorta was fairly narrow, measuring 6 cm. in circumference. More striking, however, was the marked narrowing just beyond the origin of the left subclavian artery at the point of junction with a very prominent ligamentum arteriosum, which measured 0.6 cm. in thickness and 1.0 cm. in length (Fig. 3). Here the aorta measured 0.5 cm. in diameter for almost 1 cm. Internally, there was a small channel measuring 0.2 cm. in diameter through this narrowed portion.

Beyond the point of narrowing the descending portion measured 3.7 cm. in circumference. The innominate, left common carotid and left subclavian arteries were almost twice the normal caliber. Similarly, the intercostal arteries were two or three times the normal caliber, and a small kinking with associated notching of the ribs was noted laterally in the chest wall. There was thickening of the walls of the larger arteries at the base of the brain, but no atheromatous

FIGURE 3. *Gross Specimen of the Great Vessels, Showing Marked Narrowing of the Aorta Just Beyond the Prominent Ligamentum Arteriosum.*

plaques. The remainder of the organs were not especially noteworthy.

Several microscopical sections of the myocardium through the interventricular septum just below the aortic valve in the region of the bundle of His failed to reveal the bundle but showed considerable interstitial fibrosis and coarse myocardial fibers with no inflammatory infiltration. An increase in interstitial fibrous tissue was also noted in the myocardium at the base of the mitral valve. This did not extend into the valve leaflet, nor were similar foci found elsewhere in the myocardium.

Sections taken horizontally through the origin of the abnormal commissure from the aorta and stained for elastic tissue revealed the annulus fibrosus superficial to the media, in the same relation as a normal commissure (Lewis and Grant[10]). However, a coronal section taken through the rudimentary ridge just before it joined the aortic cusps and stained for elastic tissue revealed a single delicate elastic lamina running immediately beneath the endocardium, without evidence of fibrosis or inflammation.

The lung sections showed small, patchy areas of atelectasis, and the alveolar walls appeared slightly thickened. Frequent areas of large confluent alveoli containing septal remnants with bulbous tips were present.

There was moderate sclerosis of the small arteries within the substance of the brain, especially in the gray matter. In the white matter, in addition, there were scattered phagocytes containing brown hemosiderin pigment in the perivascular spaces of some of these vessels.

The pathological diagnoses were coarctation of the aorta; bicuspid aortic valve; calcific aortic valve with stenosis and insufficiency; cardiac hypertrophy and dlatation; auriculoventricular block, complete, clinical; interstitial fibrosis of the myocardium in the region of the bundle of His; fibrous pericardial adhesion; acute passive congestion of the liver, spleen and intestines; peripheral edema of the lower extremities; and pulmonary emphysema.

Discussion

There was little doubt from the first that this patient suffered primarily from coarctation of the aorta and heart block. The chief problem was the interpretation of the murmurs and the explanation of the cause of the heart block.

In the present day of surgical correction for coarctation, the interpretation of murmurs becomes increasingly important as an indication of possible complicating lesions. The classic murmur of coarctation is described as a basal systolic murmur of slight to moderate intensity, with radiation to the upper dorsal spine. However, numerous variations have been reported, from complete absence of murmurs to a systolic murmur heard only posteriorly. The presence of associated diastolic murmurs at the base has been mentioned in various articles. Reifenstein et al.[2] stated that diastolic murmurs were present in 20 of their 104 cases, in each of which, where the valves were described, there was either aortic-valve disease or a patent ductus arteriosus. In a clinical series of 40 cases of coarctation observed by one of us (R. J. C.),[14] basal diastolic murmurs were heard in 14, and an additional 5 showed this finding on phonocardiography, giving a total of 47 per cent. Reifenstein and his associates[2] found bicuspid aortic valves in 42 per cent of their series. It has been suggested that in the presence of coarctation with high pressure in the proximal aorta, bicuspid aortic valves may give rise to a diastolic murmur. Although this relation has not been proved, the correlations are suggestive. Rheumatic valvular disease in association with coarctation is not unusual, being present in 20.4 per cent of the series reported by Reifenstein et al.[2] Apical murmurs may be secondary to the coarctation or due to separate mitral-valve disease.

In the case under discussion there was a suggestive history of rheumatic fever at the age of ten. We first considered that accompanying rheumatic aortic valvulitis might account for the basal diastolic murmur, but because of the frequency of associated bicuspid aortic valves this diagnosis was also postulated. The basal systolic murmur was considered as arising from the coarctation. The murmur radiated into the neck, which is not uncommon in this condition. The aortic second sound was present and no thrill was made out. For these reasons a clinical diagnosis of aortic stenosis was not made.

Because of the heart block and the moderately loud systolic murmur along the sternum, the possibility of an accompanying ventricular septal defect was strongly entertained. This case and others in the literature suggest that the diagnosis may be made too often because of the presence of heart block.

The basic electrocardiographic pattern with the inverted T waves and small Q waves in Leads 2 and 3, as well as the high T waves in the chest leads, raised the question of coronary-artery disease, either congenital or acquired, as a factor in the heart block. There was no suggestion of coronary-artery pain, the T waves were not typical of coronary thrombosis, and patterns over a period of observation showed no significant change. It was thought more likely, and confirmed subsequently at autopsy, that this tracing represented left ventricular strain in a vertically placed heart.*

The degree of hypertension that developed in the last six to eight months of life is unusual in coarctation and probably in part a factor of compensation for the complete heart block. This appeared in spite of marked aortic stenosis. It is interesting that the patient complained of none of the symptoms usually referable to hypertension, possibly because of the low diastolic level associated with aortic insufficiency.

Treatment of this case was symptomatic and general. Surgical plastic repair was briefly considered, but it was thought that the obvious complications, together with the patient's age, were absolute contraindications. Sympathectomy was also discussed but dismissed, again because of the patient's condition and because of relatively unfavorable results in other less complicated cases.[14] This case demonstrated the value of epinephrine in acute phases of Adams–Stokes syndrome and the fact that such acute phases usually subside over a period of time with the clearing of block or establishment of fixed complete block.

The post-mortem examination, although explaining much of the clinical picture, also provided other problems; one was the explanation of the unusual aortic valve. Lewis and Grant,[13] in their study of congenital bicuspid aortic valves, found that the annulus fibrosus failed to break through the aortic media normally. Koletsky,[15] in a study of 18 cases described two types; in one group there was a simple bicuspid valve, and in the other there was a rudimentary congenital ridge showing some attempt to form three cusps. The case under discussion seemed to fit into the latter class. The only questionable point was the superficial position of the annulus

*It is of interest that we have recently observed 2 other cases in young men at the Massachusetts General Hospital with coarctation showing almost the identical pattern.

fibrosus at the origin of the congenital ridge. In almost all of Koletsky's cases the elastic strands of the media remained superficial to the annulus until well out in the ridge. In some the annulus fibrosus then became superficial.

Furthermore, the complete lack of inflammatory or degenerative changes in the coronal section through the rudimentary ridge seemed incompatible with acquired fusion as described by Sohval and Gross.[16] This evidence strongly favored the congenital origin of the ridge.

Transverse sections just below the aortic valve through the interventricular septum revealed only dense interstitial fibrosis, and the bundle of His was not identified. This seems to provide an anatomic explanation of the complete heart block. On the other hand, the etiology of this fibrosis remains open to considerable question.

Though the gross picture was compatible with, but not strongly suggestive of, rheumatic endocarditis and aortic valvulitis, no definite microscopical proof of this diagnosis was found. The possibility that the interstitial fibrosis was rheumatic in origin could not be ruled out; however, the lack of other foci of interstitial fibrosis elsewhere in the myocardium and the absence of additional rheumatic stigmas suggested that the local fibrosis was the result of local myocardial strain as in a case (No. 7) described by Cohen et al.,[12] which also showed interstitial fibrosis and complete heart block associated with calcareous aortic stenosis without rheumatic heart disease.

The second problem raised by the autopsy was the actual cause of death. Sudden unexplained death in cases of calcific aortic stenosis is not uncommon. Though this case did not show such severe stenosis as the usual fatal cases, the final episode was similar. The lack of any evident anatomic explanation and the known serious disturbance in the cardiac conduction system led us to attribute death in this case to sudden ventricular standstill or fibrillation.

Summary

A case of coarctation of the aorta associated with Adams–Stokes syndrome, complete heart block and a calcareous stenotic bicuspid aortic valve is presented, with post-mortem findings and a brief review of the pertinent literature.

The heart block appeared to be related to the calcific aortic valve and interstitial fibrosis in the region of the bundle of His.

This is believed to be the first case of coarctation of the aorta with Adams–Stokes syndrome and complete heart block reported.

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BALLISTOCARDIOGRAMS IN COARCTATION OF THE AORTA*

Observations before and after Operation

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THE recent interest in coarctation of the aorta can be attributed to the development of a surgical treatment eliminating the stenotic area.[1-3] In recent years the number of reported cases has steadily increased. This apparent increased incidence probably represents a new awareness leading to a much more frequent diagnosis of a condition that was formerly a medical curiosity. The incidence of coarctation as given by Levine[4] is 0.05 per cent of the population.

In large measure the diagnosis depends first upon thinking of the possibility of this condition and secondly on a few additional procedures indicated to aid in establishing a diagnosis. Coarctation of the aorta must be suspected if a diagnosis of hypertension, rheumatic heart disease or congenital heart disease is made, particularly in young persons.

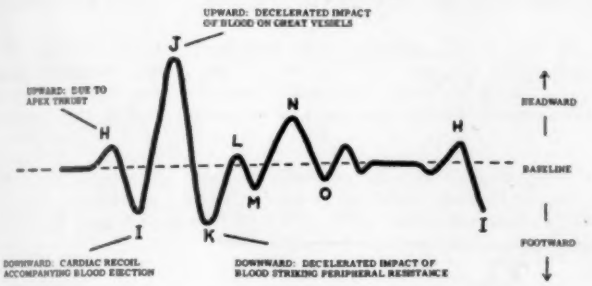

FIGURE 1. *Drawing of the Normal Ballistocardiographic Wave Pattern, with a Brief Explanation of the Most Significant Waves.*

The patient with coarctation is frequently without complaint. When symptoms are present they can be attributed to impaired circulatory dynamics resulting in an increased pressure above the stenosis. Headache and dyspnea are the two most common symptoms.[5] On occasion, palpitation, epistaxis, visual disturbances, vertigo, dizziness, intermittent claudication or angina pectoris may be attributed to this abnormality. Symptoms of heart failure or of cerebrovascular accidents are often present terminally.

Early in the course of the disease the symptoms are scant, and clinical findings more important. One should routinely feel for abdominal and femoral pulsations, which are characteristically diminished or absent in coarctation of the aorta. When this abnormality is suspected the blood pressures should be determined in the lower as well as the upper extremities to detect a reversal of the normal relation.

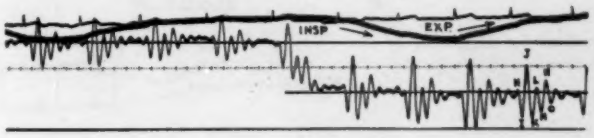

FIGURE 2. *Representative Normal Ballistocardiographic Tracing.*

Note the regularity and definitiveness of the pattern. Simultaneously recorded with the ballistocardiogram are respirations (the heavy undulating line) and the electrocardiogram. The distance between two heavy time lines is 0.2 second, and that between two fine lines 0.04 second.

Other clinical features are the demonstration of a collateral circulation, usually over the thoracic, scapular and clavicular areas; the presence of one or more cardiovascular murmurs; roentgenographic evidence, particularly that of rib erosion; and characteristic differences in direct intra-arterial radial and femoral pulsations where the femoral pulse is delayed both in the onset and in attaining its maximum height.[6]

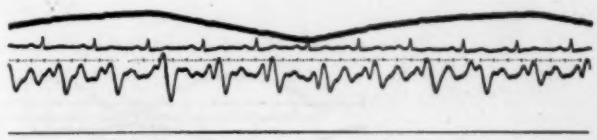

FIGURE 3. *Ballistocardiographic Tracing of a Patient with Hypertension, Demonstrating Deep JK Strokes.*

This is a common finding in hypertension.

It is the purpose of this paper to add another laboratory measurement that is characteristic of aortic coarctation — namely the ballistocardiogram. Obtaining ballistocardiographic records requires

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but a few minutes and is without discomfort to the patient. The apparatus used is an electronic device that permits simultaneous recording of the electrocardiogram, ballistocardiogram and respirations.[7]

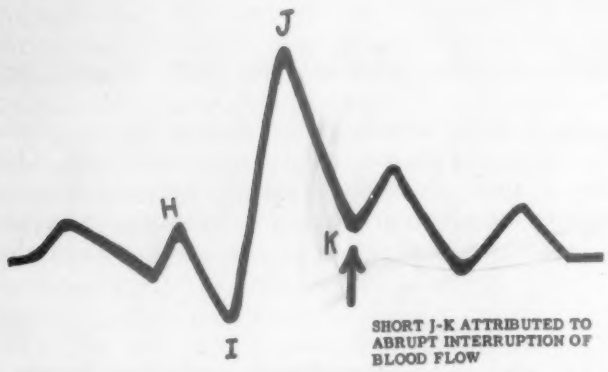

FIGURE 4. *Drawing of the Typical Ballistocardiographic Wave Pattern in Coarctation of the Aorta, Characterized by a Short JK Stroke.*

The ballistocardiogram itself constitutes a simple means of visualizing mechanical circulatory function. Figure 1 is a wave pattern as originally designated by Starr,[8] with a brief explanation of the significance of each individual wave. Figure 2 is a representative normal tracing. In coarctation the K-wave variation constitutes the single purpose of this paper. The JK stroke normally represents the decelerated impact of blood as it strikes the major peripheral arterial resistance. As would be expected typical hypertension is normally characterized by a deep JK stroke[9] (Fig. 3). The coarctation, although hypertension is present in the upper extremities, offers an early obstruction to the flow of blood through the aorta, and, again, as would be expected, this early and abrupt interruption results in an abnormally short JK stroke (Fig. 4), during and after which blood flows to the lower parts of the body primarily through the well developed collateral circulation. This was originally mentioned by Hamilton.[10]

To date ballistocardiograms have been obtained in 6 consecutive cases in which the diagnosis was firmly established, and the short JK stroke was clearly demonstrated in all (Fig. 5 and 6). If the above postulation is correct, surgical removal of the coarctation should restore normal circulatory dynamics, with a resultant typically normal JK stroke. In 1 of these 6 cases of coarctation preoperative and postoperative records that completely support the above conclusion (Fig. 7) were obtained.

The case history of this patient was as follows:

A. N., an 11-year-old boy, had been under a physician's care since the age of 4½ years, when hypertension associated with a harsh precordial systolic murmur had been discovered. These findings had persisted during the ensuing years. His only complaints were dyspnea on running, ra headaches and occasional bouts of mild epistaxis. No hi tory of coma, convulsions, paralysis, eyesight abnormaliti or genitourinary complaints was elicited. The patient w finally re-examined 1 year before operation in the hospi

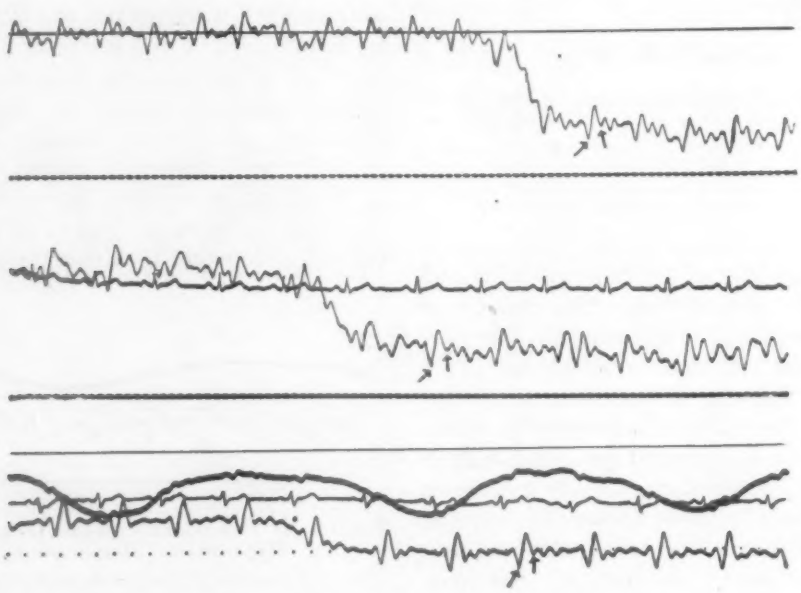

FIGURE 5. *Ballistocardiographic Records of 3 Patients with Coarctation of the Aorta in Whom the Diagnosis Was Well Established.*
Each of these tracings demonstrates the pathognomonic finding of a short JK stroke.

because of his refusal to partake in gymnasium classes at school.

The family and the past histories were noncontributory.

Physical examination revealed a slightly obese boy, in apparent good health, who tired easily upon moderate exercise. The relevant positive findings of this examination included an elevated blood pressure in both arms (right arm, 180/130, and left arm, 170/120). The blood pressures in the legs were not obtainable. A bounding radial pulse was noted, with capillary pulsations visible in the fingers. By contrast the abdominal, femoral and dorsalis pedis pulsations were greatly diminished and barely perceptible. The heart was not enlarged, with sounds of good quality. The second sounds were distinct in all areas. A normal sinus arrhythmia was present. There was a loud systolic murmur over much of the anterior portion of the chest, transmitted to the left axillary region and also to the neck. A short early diastolic murmur was heard, well localized to the apex. No thrills were palpable. The lungs were clear. No collateral vessels were noted in the usual locations.

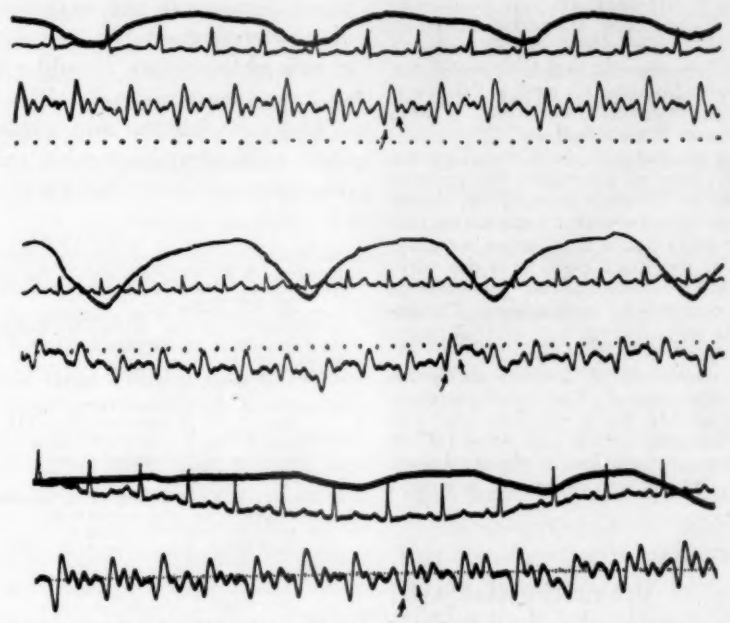

FIGURE 6. *Ballistocardiographic Tracings of 3 Patients with Coarctation of the Aorta, Showing the Short JK Stroke.*

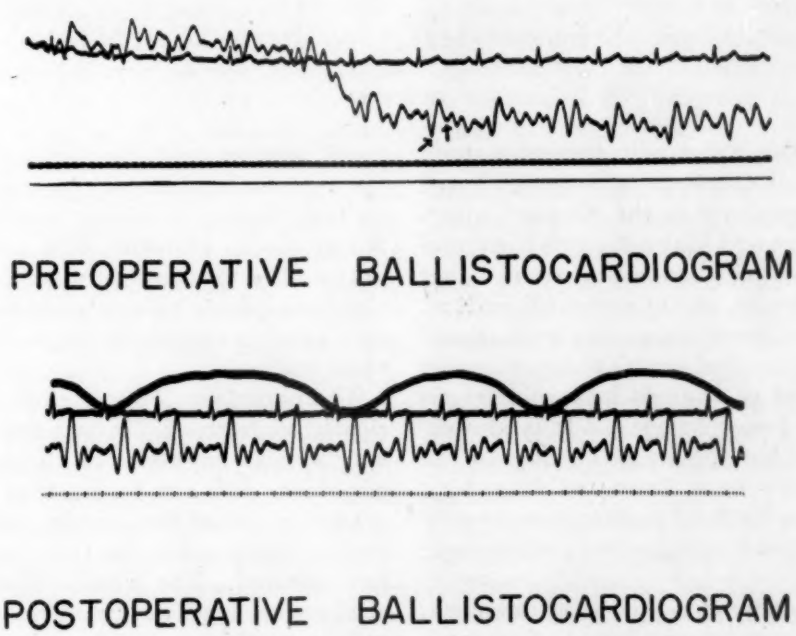

FIGURE 7. *Ballistocardiographic Tracings before and after Operation.*
Note the complete return to normal of the entire wave pattern, particularly the disappearance of the short JK stroke postoperatively. (This case is described in detail in the text.)

Examination of the blood showed a red-cell count of 4,390,000, with a hemoglobin of 12 gm., and a white-cell count of 10,000, with a normal blood smear; a blood Wassermann test and a urine examination were negative. A urea clearance test was 65 per cent and 70 per cent of normal; the blood urea nitrogen was 14.2 mg. per 100 cc., and an intravenous pyelogram was normal. A roentgenogram of the chest revealed a prominent left ventricle, but no suggestive notching of the ribs. Fluoroscopy showed definite and marked pulsations toward the neck of the vessels on both sides of the mediastinum. There was no accentuation of the ascending aorta. An electrocardiogram showed a PR interval of 0.15 second, borderline right-axis deviation, a deep S wave in Lead 1 and a diphasic P wave in Lead 3.

On March 15, 1947, the preoperative ballistocardiogram was recorded. One year later, surgical correction was successfully accomplished by Dr. R. E. Gross in Boston. At operation a point of narrowing was found about 1.5 cm. below the aortic arch. At this site the aorta had a tiny lumen, no more than 3 mm. in diameter. The narrowed portion of the aorta was removed, and by means of an end-to-end anastomosis the lumen of the aorta was restored to normal size. The patient's general condition was good during and after surgery, and he enjoyed an uninterrupted convalescence.

He has been well since operation. A ballistocardiogram taken on October 2 was entirely normal. The blood pressures at this time were recorded as 118/70 in the left arm and 124/60 in the right arm. Pressures in the legs were 140/60 in both, with a restoration of good pulsations in the abdominal aorta and femoral arteries. The heart murmurs, however, have persisted as before.

Summary

Six consecutive cases of definitely established coarctation of the aorta showed the characteristic ballistocardiographic abnormality of a shortened JK stroke. This demonstrates the usefulness of the ballistocardiogram as a diagnostic agent in suspected cases of aortic coarctation.

In the normal ballistocardiogram there is a full and uninterrupted downward JK stroke, which represents the decelerated impact of ejected blood as it strikes the major peripheral arterial resistance. The abrupt and early interruption of the flow of blood because of the coarctation, then, is the cause of the shortened JK stroke. Surgical correction of this abnormality should restore normal dynamics and produce a normal ballistocardiogram.

The case history and preoperative and postoperative ballistocardiographic tracings of a patient with this condition are presented.

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PRESENT-DAY STATUS OF FENESTRATION SURGERY*

Leighton F. Johnson, M.D.,† and Hector Silva, M.D.‡

BOSTON

THE purpose of this paper is to present a study of the results of fenestration surgery, based on our ten years' experience at the Boston University School of Medicine and Massachusetts Memorial Hospitals, on observations made at various other clinics visited and, finally, on the current literature.

In any discussion of fenestration surgery it should be remembered that the first practical, considerable operation for the relief of deafness from otosclerosis was devised by Dr. Julius Lempert. It is also interesting to observe that the major changes in the evolution of this work have emanated from him. However difficult the birth of fenestration surgery may have been, its value is now generally recognized, and its future secure.

The etiology of otosclerosis is still unknown. There is, however, an immense amount of research being carried on to determine the etiologic factors, and it appears probable that a plausible explanation for this pathologic phenomenon will soon be described. Such observers as Kobrak, Hallowell Davis, Stacey Guild, Dorothy Wolf and Weaver, working in the allied fields of histopathology, physiology and electronics, have greatly extended otologic concepts, and aural mysticism is beginning to give way to aural realism.

The pathology of otosclerosis is complex and not clearly understood. It is known that the process may appear anywhere in the bony capsule of the labyrinth. It is also known that eighth-nerve deterioration is part of the complex picture of otosclerosis, and no explanation for this phenomenon has thus far appeared. The whole subject is teeming with challenging, unsolved problems. So far, there is insufficient evidence to prove that fenestration of the labyrinth halts the progressive degenerative changes of the eighth nerve. The question whether a successful fenestration operation gives a respite from deafness for ten or fifteen years or whether it is a permanent cure cannot be answered today. Deaf-

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ened persons, however, are anxious to avail themselves of the possibility of social and economic rehabilitation even if the permanency of the operation is still in question.

The selection of cases suitable for fenestration is an exacting responsibility and plays a most important part in the success or failure of this work. To hope to restore hearing to the practical threshold, which is considered to be the 30-decibel level, presupposes a nerve response by bone conduction of 30 or better by audiometric examination and a differential between the air and bone audiogram of at least 25 decibels. This differential between the air and bone response is referred to as the cochlear potential. The tuning forks continue to be invaluable in the diagnosis of otosclerosis and in the selection of suitable cases for operation.

It is true that certain patients with an audiometric nerve response in one or more frequencies below 30 decibels improve remarkably after operation. In many such cases both the patient and operator are conscious of the probability of failure, and occasionally there is a gratifying result. However, we consider the favorable response too infrequent to justify the procedure except in rare cases.

The principle behind the fenestration operation is a delightfully simple one. In brief, the oval window has been invaded by the otosclerotic process, thereby partially or completely shutting out the airborne sound waves from the labyrinth. The operation calls for the construction of a new window into the labyrinth through which the sound waves may pass. The execution of this simple principle to the ultimate goal of restored hearing is, however, complex and highly technical.

The technic of the fenestration operation is familiar to all. Its evolution embraces two main eras. During the first period, in which the window was made in the external horizontal canal, the incus was left in situ. The immediate results of this operation were spectacular; however, within the first year two thirds of the cases had bony closure of the fistula, and the hearing dropped to the preoperative level. In the present era the fistula is made in the surgical dome of the vestibule, and the incus is removed. This new window has been named the novovalis. In this location a much larger fistula can be made, and the selection of this site by Lempert doubtless saved fenestration surgery from the discard.

Innumerable attempts have been made to influence the osteogenic reaction around the fistula by a wide variety of procedures ranging from the gold burr, various obturators and cartilaginous stopples to the lead burr. Lempert has proved by experiments on monkeys that the lead burr definitely inhibits osteogenesis. It is now our practice to use the lead burr routinely. The present technic of preparing the fistula by removal of the lid in one piece obviates the production of much bone dust and thereby lessens the likelihood of bone regeneration.

All aspects of the fenestration operation must be performed with technical finesse, but it is the fistula that requires meticulous endeavor, and success or failure largely hinges on this aspect of the operation.

We believe that progress has been too rapid in the endeavor to handle the difficult problems connected with osteogenesis and fistula patency. Certain procedures that appeared sound from the standpoints of physiology and physics have sharply reduced the percentage of successes — for example, the cartilaginous stopple and the use of the dental excavators. Most operations in which the stopple was employed did not bring the hearing acuity to the practical threshold. The use of the dental excavators stimulated the endosteal layer, with resulting untoward osteogenesis. We are convinced that the present cupola technic of Lempert or the double line procedure of House will give a sharply increased percentage of success in cases reported one or two years hence.

The causes of failure of the fenestration operation include improper selection of cases and bony closure of the fistula. The incidence of bony closure when the fistula is made in the surgical dome of the vestibule by the cupola or twin line technic and properly treated with lead has been reduced to approximately 10 per cent. It should be emphasized that the flap should undergo most careful scrutiny under magnification for the presence of bony spicules. Obviously, if a bony spicule is attached to the undersurface of the flap and the spicule falls over the fenestra, early closure is certain. Revisions when bony closure has reduced the hearing to the preoperative level are far more successful today than in the era of the fistula in the external horizontal canal.

Aseptic labyrinthitis continues to be a possible source of failure. However, with penicillin given routinely for five days, its incidence has been greatly curtailed. Great care must be exercised in irrigation so that the membranous labyrinth is not traumatized while bone dust is being washed away from the endosteum; any instrumentation calls for meticulous effort to avoid trauma to the labyrinth.

Thermal changes to the labyrinth by excessive heat caused by the polishing burr or an irrigation solution that is too warm must be zealously guarded against.

There must be complete hemostasis at the time the labyrinth is opened.

The anesthesia for the fenestration operation seems to vary with each clinic. The following is the anesthetic procedure in use at the Massachusetts Memorial Hospitals:

Preoperative medication should be adequate to provide a good night's sleep and to present the patient in the operating room quite drowsy and euphoric: 2 gm. of phenobarbital at bedtime, the

dosage being repeated if necessary for sleep; 2 gm. of phenobarbital by mouth at 6 a.m.; 2 to 6 gm. of neonal by mouth at 6:30; 5 to 8 mg. of morphine sulfate subcutaneously at 7; and 5 to 8 mg. of morphine sulfate and 0.6 mg. of atropine sulfate subcutaneously at 7:30.

On arrival in the operating room at 7:45, a needle is inserted in a vein, preferably of the lower extremity, and an infusion of dextrose in water is started. The pharynx is sprayed with 2 per cent pontocaine. Sodium nembutal, in a dosage adequate to induce sleep, is injected into the tubing (50 to 150 mg. is usually sufficient). A continuous infusion of 0.4 per cent pentothal sodium is started about five minutes before incision by the insertion of a needle through the tubing so that the solution runs in through the needle previously placed. When the patient is sufficiently relaxed, a Guedel oropharyngeal airway is placed and oxygen is run in through a catheter threaded through this during operation. The total dose of pentothal sodium is usually 1 gm. or less, and rarely is as much as 1.5 mg.

This anesthesia, in the hands of the Anesthesia Service, has proved safe and satisfactory.

Postoperatively, each patient receives 300,000 units of penicillin daily. Pyribenzamine, in amounts of 50 mg., is given four times a day. This is administered empirically in the hope that its antihistaminic action may prove beneficial in thwarting the possibility of chemical labyrinthitis. The cavity is packed with either paraffin gauze or small sea sponges. We have been using the sea sponges for some time, since they are easy to apply and remove, and are successful in firmly attaching the flap to the bony surface. The packs are removed on the fifth day under careful aseptic technic, and the patient leaves the hospital on the tenth day. Office dressings are done once or twice a week thereafter until the cavity is dry and epidermatized.

Throughout our entire series we have had no mortality and no injury to the facial nerve. There have been no untoward complications such as lateral sinus thrombosis and intracranial disease The complications have been limited to local allergic reaction to the sulfonamides; a few cases of persistent aural discharge requiring considerable time and patience to dry up; a case of acute otitis media after operation, with spontaneous rupture (with large doses of penicillin the middle ear became dry, and the perforation healed with no ultimate damage to the improved hearing); and 4 cases of atresia of the external auditory meatus, requiring excision of scar tissue.

The results of fenestration surgery may be divided into four groups as follows: patients who show a postoperative hearing level of 30 decibels or better for the vital frequencies of 512, 1024 and 2048; patients whose postoperative hearing level is not sustained at the 30-decibel level but in whom definite gain in hearing acuity has taken place; patients whose hearing remains at the preoperative level; and those whose hearing has become worse after operation.

Table 1 shows our record at the Massachusetts Memorial Hospitals with the novovalis operation from 1942 to 1946. Any skillful operator can have an immediate record of at least 80 per cent successes, but at least one year should transpire before the permanency of the hearing can be accurately evaluated. It should be mentioned that this list includes 4 cases in which the stopple was employed, none of which reached the practical threshold. Many of the patients listed as improved are perfectly rehabilitated, but since their audiograms do not show the practical threshold of 30 decibels, they are labeled only as improved. Nearly all in this group had practical hearing immediately after operation, but in the year and a half since then, 14 per cent have fallen to the preoperative level. Many are suitable for revision.

TABLE 1. *Results of Novovalis Fenestration Operation in 151 Cases, 1942–1946.*

RESULT	PERCENTAGE OF CASES
Practical hearing	60.0
Improved hearing	26.0
Preoperative hearing level	13.0
Hearing worse after revision	0.88

TABLE 2. *Results in the Experimental Group (Diminished Bone Conduction), 1942–1946.*

RESULT	PERCENTAGE OF CASES
Practical hearing	31
Improved hearing	18
Preoperative hearing level	51
Hearing worse	0

The second group comprises 32 experimental cases in which one or more of the vital frequencies were below the 30-decibel level by bone conduction (Table 2).

CONCLUSIONS

We believe that no patients with otosclerotic deafness have experienced restored practical hearing except by the Lempert fenestration operation. Several thousand patients today have been socially and economically rehabilitated by this means. In skilled and trained hands the surgical hazards are minimal. The likelihood that further impairment in hearing will result from operation is less than 1 per cent.

The record of the last two years gives every promise of substantial improvement in the per-

centage of practical hearing, because of the improved fistula technic.

The work is new and should be approached only by those with broad otologic training and an investigative temperament.

In the transition from surgical procedures for infections to operation and study for the alleviation of deafness otology has emerged increased in stature and achieved an enviable position among the surgical specialties.

MEDICAL PROGRESS

ORAL SURGERY (Continued)

KURT H. THOMA, M.D.*

BOSTON

DENTOALVEOLAR SURGERY

Dentoalveolar surgery, which includes the extraction of teeth (exodontia), probably occupies a large part of the time of most oral surgeons.

The technic of tooth extraction has been perfected. Teeth judged to be complicated on x-ray examination because of exostosis of the roots or anatomic deformity are excised by an open procedure that allows clear vision and prevents laceration of the soft tissue and fracturing of the surrounding alveolar bone.[51, 52]

Much more attention has recently been devoted to the correction of deformities and preparation of the alveolar ridge for the construction of satisfactory dentures.[53, 54] Deformities such as maxillary protrusion are eliminated by alveolectomy. Bony exostoses and hyperplasia of the gums are excised. Irregularities caused by trauma when the teeth are extracted are eliminated to produce an even ridge, and in cases of atrophy of the alveolar process, knife-edge ridges should be made smooth,[55] whereas in other cases an operation for ridge extension is indicated.[56, 57]

New methods have been developed during the last two decades for the removal of unerupted and impacted third molars. An odontectomy[58] is performed if the tooth is impacted. This includes tooth division to preserve the bone, large parts of which formerly had to be removed to dislodge the tooth *in toto*. Pell and Gregory[59] report their findings on the basis of a ten-year study and recommend tooth sectioning highly because of the small amount of operative trauma, which reduces the postoperative discomfort.

It is generally believed that the removal of these malposed teeth can be more easily performed in young persons, and others have gone so far as to recommend prophylactic odontectomy[60, 61] to prevent the crowding and disalignment of anterior teeth. Such malocclusion is caused by pressure of the third molars in their attempt to erupt. Other reasons for the early removal of these teeth are, according to Gunter,[62] the following: their eruption may be accompanied by pain and infection of the investing structures (pericoronal infection); they are often hypoplastic and susceptible to dental caries; their repair, if decay is present, is difficult and inadequate; and they tend to disturb the alignment of other teeth. In addition should be mentioned obscure, referred pain, which may radiate to other branches of the fifth nerve or may even involve other nerves of the face and head.[63] There is no room in the modern dental arch for third molars, and few of them ever become functional teeth.

The patient's general health and resistance should be considered in case of multiple extraction of infected teeth, and the dentist should consult with the patient's physician.[64] This is especially true in cases of diabetes and cardiac, thyroid and renal diseases. Most patients with such diseases should be hospitalized, properly prepared and premedicated, and given the protection of antibiotic therapy before and during the operation and also postoperatively. Kane[65] points out that the danger of bacteremia during the extraction of infected teeth should always be kept in mind. The risk of bacterial shower is present not only in apical abscesses but also in periodontal infection. O'Kell and Elliott[66] have recovered *Streptococcus viridans* from the blood stream in 75 per cent of 40 patients suffering from marked pyorrhea. Barnfield,[67] studying autopsy records of 92 cases of subacute bacterial endocarditis, found in 6 a relation between the disease and extraction. Of 40 reported cases supposedly precipitated by dental extraction, in 15 there was convincing evidence, whereas in 10 others the relation seemed probable. Kane[65] believes that oral surgeons may do much to prevent the occurrence of subacute endocarditis by giving patients with heart murmurs prophylactic penicillin, which is superior to the sulfonamides in preventing transient bacteremia. Pressman and Bender[68] report an incidence of bacteremia as high as 80 per cent after

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tooth extraction. The American Heart Association[69] has urgently recommended that dentists take advantage of the prophylactic effect of penicillin when extracting teeth for patients with rheumatic heart disease.

Patients with a hemorrhagic diathesis require special consideration. Hemorrhage following tooth extraction has always been feared though lately great progress has been made in its prevention. In many cases, local causes are present, such as fractured alveolar process and lacerations of the gingiva. If bleeding is due to systemic disease, a more serious situation exists, and general as well as local therapy is indicated. The patient should first of all be hospitalized, and a thorough blood study should be made. The following treatment has been recommended.[70]

In ascorbic acid deficiency, which is generally recognized because it produces a typical scorbutic gingivitis besides the well known general signs, spontaneous hemorrhages occur in the mouth from the bluish-purple, bloated gums. Patients should receive 500 mg. of ascorbic acid per day with vitamin P (citrin) to decrease the capillary fragility.

In thrombocytopenic purpura, splenectomy may almost immediately cause a marked increase in the platelet count, and fresh blood transfusions are valuable. In cases of secondary purpura that are not improved by splenectomy in addition to transfusions, the use of moccasin venom has been recommended.[71]

In cases of pernicious anemia, liver therapy is indicated; in cases of hemorrhage associated with obstructive jaundice, vitamin K with bile salts should be given. In the leukemias, blood transfusions to decrease the danger of hemorrhage in preparation for necessary tooth extraction, which should be avoided if possible, are considered helpful. In hemophilia, a cure cannot be expected, but in anticipation of tooth extraction, or if the patient has started to bleed spontaneously, transfusions of fresh blood (twelve to twenty-four hours old) are recommended to reduce the coagulation time. Small amounts of blood (50 to 100 cc.) will reduce the coagulation time markedly. Such transfusions should be repeated frequently until the danger of hemorrhage has passed. They should be given preoperatively, during the operation and postoperatively.

Lewis and his associates[72] reported that normal plasma contains a substance that is absent in hemophilic plasma and is a subfraction of the active globulin fraction with which it is associated. In the Plasma Fractionation Laboratory of the Department of Physical Chemistry, Harvard Medical School, an antihemophilic globulin, Fraction I, of normal blood plasma has been developed for intravenous administration and is provided by the Red Cross without charge. It will reduce the coagulation time to about fifteen to thirty minutes after one or two intravenous injections of the content of two vials containing 200 mg. each, diluted in 5 cc. of sterile water. The tooth extractions should be performed as soon as the clotting time has been reduced to safe ranges, and it is recommended that all the teeth that have to be extracted be removed at one sitting. The reduction of the clotting time persists for about four hours, after which it slowly rises to its previous high level. The injection may be repeated until the wound has healed completely. This treatment, however, has not been found to give uniformly good results, and blood transfusions may have to be given as well. It is believed by some hematologists that there is another factor lacking in hemophilia, such as a serum activator, that has not been discovered as yet.

Other patients may have a predisposition to hemorrhage that is not so easily recognized. Among these are patients with prothrombinopenia, which may arise from lack of vitamin K because of dietary deficiency, faulty absorption due to lack of bile salts (obstructive jaundice, biliary fistula and sprue) or severe infection and liver damage. Here should be mentioned the effect of the prolonged use of acetylsalicylic acid, which may decrease the prothrombin. Prothrombinopenia may be corrected in such cases by the administration of vitamin K. Cahn[73] recommends that 1 mg. of vitamin K be prescribed to every 0.38 gm. of aspirin, if the drug is taken constantly.

There is also recognized nonhemophilic hereditary hemorrhage, which occurs in patients with perfectly normal hematologic findings, except the bleeding time, which is prolonged. They suffer from prolonged bleeding after injuries, especially the extraction of teeth. In some the tourniquet test is positive whereas the platelet count and coagulation times are normal. Offspring of either sex may inherit the disease. Levy[74] recently reported 4 such cases with prolonged bleeding after extraction of teeth.

Local Treatment of Hemorrhage

Before the local treatment is described, it should be pointed out that patients with or without hemorrhagic diathesis who have been bleeding excessively become dehydrated. Fluids should be forced by mouth or given intravenously, or blood should be given if the hemoglobin and red-cell count are low.[75]

After the extraction of teeth, meticulous care should be taken to arrest all bleeding from soft-tissue wounds. Often suturing is indicated. A sterile pack of gauze may be applied between the jaws. It is placed on the bleeding socket or on an oozing wound that has been sutured, until the clot has formed. Excessive bleeding from a tooth socket may be dealt with by the placing of thrombin or

oxidized cellulose into the alveolus, as described below.

In case of secondary hemorrhage after tooth extraction the socket should be debrided, all old clot removed, and a pressure pack applied. The patient should be placed in a semi-Fowler position if in bed, or should be directed to sit in a chair rather than to lie down, to decrease the blood pressure in the head. Cold compresses applied to the region or to the neck are often useful. Rinsing the mouth should be prohibited.

Various drugs have been recommended for local application. Epinephrine in 1:1000 solution is generally employed during operation. The use of ferric subsulfate powder pressed into a socket with iodoform gauze and left for several days has given good results, although the black precipitate is objectionable. In patients with hemorrhagic diseases the bleeding may be arrested by the local application of fresh raw meat, fresh blood or cephalin. Beef or rabbit globulin can be applied on gauze into the socket, and has been recommended as a great aid in such bleeding after tooth extraction. Today, oxidized cellulose[76] may be left in the wound permanently, avoiding the danger of new hemorrhage, which is always feared when a medicated gauze pack is being removed. I have found thrombodent cones,* which contain 1 unit of thrombin and 1 mg. of tyrothricin, extremely effective; they are ideal for use in the mouth because they contain an antibiotic that is effective against gram-positive and some gram-negative organisms found in the oral cavity. One to three are placed into a tooth socket, or if the bleeding occurs at the gingival margin, they can be crushed to a powder and held in place by an intermaxillary pack. These local measures are of great help whether or not general medication and blood transfusions are used.

Odontogenic Infections

Focal Infection

Chronic dental abscesses and periodontal diseases as foci of infection played an important role three decades ago. Today focal infection is not heard of so much in this country, although abroad papers on this subject are making up a considerable part of the literature. This may be due to the great clean-up that followed the denunciation of septic dentistry by William Hunter in his famous inauguration lecture at McGill.[77] The American dentist was quick to change his attitude toward the retention of all teeth whether good or bad as long as they were asymptomatic, and discarded practices that led to the formation of abscesses. When it is deemed advisable to retain a tooth in spite of an infected pulp, root-canal therapy is undertaken. Bacterial tests are made during the treatment of the root canals, which are not filled until they are free of infection.[78]

Reimann and Havens[79] concluded that the field of focal infection has been definitely narrowed from former concepts. Several sobering articles have appeared to discourage those who believe that all ills can be cured by the extraction of teeth, and it has been pointed out that the removal of a focus of infection presents more prophylactic than therapeutic potentialities.[80] However, the theory of focal infection should not be entirely discredited since there are still occasional cases in which there is a good probability of the relation of infected teeth to the systemic disorder of the patient. Kravitz[81] believes that, of all foci of infection, those of the teeth are most closely related to the eye. Slocumb et al.,[82] of the Mayo Clinic, discussing the effect of the removal of foci of infection in heart disease, chronic infectious arthritis and glomerulonephritis (conditions representative of those in which removal of foci of infection has been said to be of clinical benefit), state that in heart disease focal infection can be considered to be a factor only in an occasional case of coronary thrombosis or acute pericarditis. However, they lay stress on the importance of protecting patients by means of chemotherapy when septic tonsils and badly infected teeth are removed in the presence of an old valvular lesion. As previously mentioned, bacteremia following dental extractions may become the starting point for acute bacterial endocarditis. In chronic infectious arthritis, the authors recommend eradication of a definite focus if present after the progression of symptoms is under control. Chronic glomerulonephritis is considered to be caused by repeated and continued toxic assaults on the kidneys originating in acute and chronic foci of infection. Patients with chronic nephritis, intermittent suffering and exacerbations have more than 80 per cent evidence of chronic infections about the roots of teeth, tonsils or sinuses, and clearing up such foci not only shortens the convalescent period of the acute episode but also prevents further damage to the kidney. Ensign[83] points out that infected pockets in periodontal disease are an important focus, and the mobility of the teeth aids toxic absorption, "the tooth acting as a piston pumping bacteria into an area of rich capillary supply."

Acute Alveolar Abscesses

These inflammations and their sequelae, deep infections involving the fascial planes around the jaws, have been accompanied with very serious complications. Keefe[84] called attention to the fact that three periods of disease may be distinguished: that in which the initial lesion develops, that of extension to the fascial spaces, and that of serious complications through embolism, pyemia and septicemia. Haymaker,[85] of the Army Institute of Path-

*Available from L. D. Caulk Company, Milford, Delaware.

ology, has described 27 cases of fatal intracranial complications of dental infection. Direct spread was more often caused by maxillary than by mandibular teeth, whereas in hematogenous infection the reverse was true. Osteomyelitis of the sphenoid bone occurred in 8 cases, brain abscess in 7, cavernous-sinus thrombosis in 9, infraorbital abscesses in 6, and intraorbital abscesses in 3. Hargraves[86] stresses the need for early and adequate treatment to reduce the mortality by preventing such complications.

The best treatment of acute dentoalveolar abscesses is prompt extraction of the tooth, under the protection of antibiotic therapy. A culture should be taken to establish the penicillin sensitivity of the organisms. Swelling of the face and early cellulitis generally require no treatment besides continuation of parenteral administration of the antibiotic.[87] In advanced cases when submaxillary, submental, sublingual, parapharyngeal, masseter, parotid and infratemporal abscesses or their complications have already developed, removal of the original focus (tooth) will not afford sufficient drainage, and the antibiotic treatment will not be effective without operative interference.[87] Incision and drainage should be resorted to, and great care should be taken to open deep accessory abscesses. Since penicillin has come into use, complications are less frequent and less likely to have a fatal result. An example is a case reported by Wiesenfeld and Philipps[88] of a patient with thrombophlebitis of a cavernous sinus developing after tooth extraction in spite of adequate sulfadiazine administration, who made a dramatic recovery after the administration of penicillin (150,000 units a day). Ludwig's angina also has a much better prognosis, since penicillin has given striking results in early cases with and without incision.[89]

Bacteriologic examination is of great importance since mixed infections are very common in the mouth. Colon bacilli, gram-negative organisms and especially the fusospirochetal organisms are frequently found accompanying infections by the streptococcus or staphylococcus, and in some cases the use of streptomycin is therefore indicated. Actinomycosis frequently yields to penicillin therapy. Nichols and Herrell[90] reported cures in 24 out of 26 cervicofacial cases. They recommend the administration of 500,000 units daily for six weeks.

Osteomyelitis of the Jaws

The treatment of osteomyelitis of the jaws, which may be caused by dental infections and septic fracture, occasionally by furunculosis[91] and through hematogenous transportation of bacteria,[92, 93] has also undergone a considerable change since the advent of chemotherapy.

Infantile Osteomyelitis of the Maxilla

This condition has a better outlook. Intramuscular injections of penicillin, 80,000 to 800,000 units a day, are recommended and, if an intraoral fistula is present, should be supplemented with penicillin irrigations.[94] Schenk[95] described 5 patients all of whom recovered in spite of a mortality of 20 to 50 per cent. He recommended that penicillin should be administered promptly, and a minimum of 800,000 units should be given over a long period. Sometimes, penicillin combined with sulfonamide is recommended in mixed infections.

Osteomyelitis of the Jaws in Adults

This inflammation occurs in an acute fulminating form and the more frequent subacute and chronic types. The former is often aborted if penicillin treatment is instituted even before x-ray evidence of bone changes can be demonstrated.[96] In most of the subacute and chronic cases the use of antibiotics and the removal of the cause do not result in resolution without operative interference to eliminate pus and necrotic tissue since patients frequently do not seek treatment until extensive suppuration and bone necrosis have occurred.[87] Various operative procedures performed under antibiotic therapy are recommended,[97] including surgical drainage, sequestrectomy and saucerization, fixation in pathologic fracture and very rarely excision of the affected segment of bone. Sequestrectomy is simple if an intraoral approach is possible; frequently, however, the sequestrum is encased or inaccessible, and an extraoral sequestrectomy is recommended.[98] This is combined with careful débridement and saucerization of the bone defect. Excision (condylectomy) is especially indicated if the infection is restricted to the condyloid process, which occurs as a complication of an infratemporal abscess, since osteomyelitis here generally causes partial or complete ankylosis of the mandibular joint.[99] In any of these procedures intramuscular administration of antibiotics should be supplemented by local instillation of a solution of 1000 to 5000 units of penicillin per cubic centimeter into the involved area, as recommended by Smith-Petersen, Larson and Cochran.[100] A rubber catheter sutured to the margin of the incision gives satisfactory results and allows primary closure of the wound.

The determination of the bacteria causing the infection is recommended in each case, with their sensitivity to specific antibiotics. Although staphylococci and streptococci are the common bacteria found, Vincent's organism, colon bacilli and many other aerobic and anaerobic organisms may be encountered. It is well to have a serologic test because syphilis occasionally simulates pyogenic osteomyelitis,[101] and in the localized form tuberculosis should be kept in mind.[102] Pathological examination of the excised tissue establishes the diagnosis.

Good postoperative care is of great importance in osteomyelitis. This includes a liquid diet high in protein, caloric and vitamin content. The adminis-

tration of fluids and electrolytes is indicated, and the use of blood transfusions to improve the patient's general resistance aids the recovery.

Irradiation Osteomyelitis

Necrosis of the jaw, as well as a slowly progressing sclerosing type of osteomyelitis, is produced by heavy irradiation for the treatment of cancer. The resorptive and regenerative power of the bone is destroyed. Watson and Scarborough[103] reported on 235 cases of osteonecrosis found in a group of 1819 patients with cancer of whom 12 died free of cancer as a direct result of jaw infection. Colby[104] makes a statement with which I thoroughly agree — namely, that radical dental treatment is indicated before irradiation, all remaining teeth being extracted. Irradiated patients on the other hand should have no operative procedures on the irradiated jaw for years. If teeth remain, proper hygienic conditions should be maintained, and extremely conservative dental treatment is imperative. The use and construction of protective prostheses containing lead shields has been described by Ackerman.[105] They are used to protect the regions of the jaw that are not to be irradiated.

Fractures of the Maxilla and Mandible

Great progress was made in the treatment of fractures of the jaws during World War I, but there were certain limitations, which were only recently overcome with the advent of the use of antibiotics. Simplified methods of fixation that require less technical work (dental splints) have been developed,[106] assuring more rapid results with less discomfort to the patient. This refers particularly to transosseous wiring and skeletal fixation.

Transosseous Wiring

While the use of open reduction with internal wiring fixation has been discouraged in the past, Gordon[107] reported the use of this method for 7 malar, 2 maxillary and 15 mandibular fractures. Adams[108] described a method of fixation for maxillary fractures by wiring the fractured parts to neighboring unfractured bony structures to avoid uncomfortable extraoral appliances such as headbands and plaster caps. The prophylactic use of penicillin and of stainless steel or tantalum wire, which are biologically inert, probably plays a great part in the recent success with these methods. It is, however, important not to rely on the wires for the immobilization of jaw fractures. The wire should serve only to hold the fragments in the reduced position so that they cannot be displaced by muscle pull. The fracture should be fixed by immobilization of the mandible.[109]

Indications for transosseous wiring are mandibular fractures at the angle of the jaw with upward or lateral displacement of the posterior segment, fractures through an unerupted third molar, which should be removed, and comminuted fractures with small displaced segments.

Transfixation with Steinmann Pins

This method is hazardous because it easily causes injury to the inferior alveolar nerve and vessels, resulting in anesthesia of the lip and intraosseous hematoma. However, it is useful[110] in fractures of the symphysis, as well as for craniomandibular fixation. This method and others eliminating the use of headbands and plaster caps were described by Thoma.[111]

Skeletal Pin and Clamp Fixation

Numerous advantages are claimed for this method. Winter,[112] in a report on 36 cases, stated that skeletal fixation made early and prompt reduction possible, since it required a minimum of complicated equipment and therefore could be applied in dressing stations, field hospitals, evacuation hospitals or on shipboard. Of first importance in war surgery, he stated, was the fact that with this method the soldier with a mandibular fracture may be transported after the fracture has been reduced without danger to his life from sea or air sickness. Secondly, because the bone is immobilized but the joint is free, moderate function is possible, which is a great advantage not only because it provides better nourishment for the patient, eliminating the preparation of liquid diets, but also because it favors healing. Haynes[113] pointed out that callus formation may be expected earlier because it is known that the use of a bone increases the inflowing calcium and counteracts the atrophy that occurs from disuse. Other writers pointed out additional advantages; Mowlem et al.[114] stated that the method facilitated oral hygiene, simplified feeding and prevented stiffness of the joint and muscles. Griffin[115] added that drainage, inspection and irrigation were easily made in compound fractures and fractures with extensive wounds. Gillies[116] stressed the perfect control of the fragments into which the pins were inserted and the easy anatomic positioning.

The indications for the use of skeletal fixations were set down by Waldron.[117] He recommends the method for badly displaced fractures of the edentulous mandible, particularly when they are comminuted; fractures behind the angle of the jaw, when the posterior fragment becomes difficult to maintain in reduced position; multiple fractures involving both the upper and the lower jaw, when other methods are not satisfactory because of the loss of teeth; comminuted fractures of the mandible with destruction of a section; and cases in which facilities for construction of dental splints are not available.

Thoma[118] found skeletal fixation unsatisfactory for cases of fractures at the angle of the jaw; pins will not hold well or last, because the bone in this

part of the mandible is very thin. He developed a peripheral bone clamp that can be placed around the jaw through a suitable incision in the skin, which is closed. Only the pin protrudes through the skin and serves as an attachment for the connecting bars of the fracture appliance fastened to half pins inserted in the anterior fragment.

Bradford and Wilson,[119] discussing skeletal fixation, give two good rules to prevent misuse or abuse of the method: the apparatus should be applied only by an experienced surgeon who thoroughly understands the principles of its use and who has carefully studied its mechanism; and the procedure should not be used routinely but should be applied only in cases in which treatment by simple, conservative methods seems to be inadequate.

Fractures of the Mandibular Condyle

These fractures, treated in the past by immobilization of the mandible without any attempt at reduction, have not given such uniformly good results as some of the proponents of conservative treatment suggest. In cases of gross displacement or fracture dislocation this treatment frequently results in deformity, functional debility and malocclusion, with deviation on opening of the mouth, and even ankylosis may occur.[120-122] A method of open reduction and wiring fixation is recommended to prevent these deformities.[122] If they do occur, condylectomy will have to be resorted to to give relief.[121]

In conclusion, it may be said that the treatment of jaw fractures requires a great deal of experience with a variety of methods old and new. In a discussion of the history of the treatment of jaw fractures[123] some of the principles regarded as essential have been set forth and discussed. These are as follows: treatment of the patient; attention to soft-tissue wounds; carefully performed clinical and x-ray examination; determination of the type and exact location of the fracture; prevention and treatment of infection; temporary immobilization if adequate treatment has to be delayed; choice of the correct type of anesthesia; attention to proper reduction; selection of the best method of fixation and immobilization; and proper attention to aftercare.

Diseases of the Mandibular Joint

Arthrosis

Various intra-articular changes may occur in the temporomandibular joint owing either to major trauma causing injury to the meniscus or to trauma resulting from functional disturbances,[124] some of which are distinctly associated with occlusal abnormalities of the teeth. Other diseases that cause similar symptoms are the rheumatoid, hypertrophic and infectious forms of arthritis, which produce degenerative changes similar to those produced in other joints of the body, and ankylosis of the jaw may result.

Bellinger[125] describes the symptomatology as follows: the symptoms are usually preceded by a history of a snapping sound in the joint, which may occur at the beginning or termination of the opening movement and is directly related to the meniscus. It indicates hypermobility and excessive excursion. In addition, more or less profound and palpable tenderness, pain and restriction of motion may develop. Extirpation of the meniscus effects immediate cessation of the symptoms.

Other symptoms, reflex in nature, may be experienced. These are generally spoken of as the "Costen syndrome." Besides the noise in the joint during mastication, the patient complains of various forms of neuralgia, such as headaches about the vertex and occiput, pain behind the ear or in the mouth and jaws and a burning sensation or pain in the tongue. Wright[126] pointed out that deafness may occur; vertigo and tinnitus (noises, buzzing and roaring) may be present, according to Goodfriend.[127] The dental signs are unusual attrition or loss of or incorrect replacement of the posterior teeth.

The treatment in many cases may be conservative, consisting of resting the jaw and the use of heat. Rest may consist of the omission of food that requires force to masticate, or intermaxillary wiring for complete immobilization. Heat may be applied by means of flaxseed poultices or diathermy. In some cases restoration of the occlusion or wearing of a splint to open the bite and distracting the joint surfaces give good results.[128] Operative treatment consists of injection of a sclerosing solution, menisectomy and condylectomy or osteoarthrotomy.

Injection

Injection of the joint space with a sclerosing solution (sodium psylliate or sylnasol) is recommended by Schultz[129] for hypermobility, but not if the patient has acute symptoms, limited motion and pain. Clicking, grating and popping have been controlled in six to twenty-four hours.

Menisectomy

Menisectomy is indicated in recurrent dislocation, fracture or tear of the meniscus due to trauma, partial erosion of the meniscus in traumatic arthritis, blocking of the condyle and noise and cracking not relieved by conservative treatment.[130] Boman[131] reported 58 cases of treatment by excision of the meniscus.

Condylectomy and Osteoarthrotomy

These operations are used in more serious disease of the mandibular joint, destruction of the joint surfaces with resulting pain, as in various forms of arthritis, in partial ankylosis caused by traumatic

arthritis, malunion of condylar fracture, rheumatoid arthritis, osteomyelitis of the condyle and hypertrophy (osteoma) of the condyle and true ankylosis. The causes of ankylosis[99] are birth trauma, rheumatoid arthritis, including Marie-Strümpell disease, suppurative arthritis, osteomyelitis of the condyle and fracture of the condyle with hemarthrosis.

Dingman[132] and Thoma[99] prefer osteoarthrotomy to arthroplasty since there is less chance of recurrence because of the large separation of the condylectomized ramus from the glenoid fossa or temporal bone (1.5 to 2.0 cm.). Hickey[133] has experimented with oxidized cellulose (oxycel), which he packs into the space not only because it controls the bleeding but also because it is said to interfere considerably with bone repair.[134]

(To be concluded)

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 35181

PRESENTATION OF CASE

First admission. A fourteen-year-old girl entered the hospital complaining of pain in the left hip for eighteen months.

Ten months before admission the patient had an attack of "streptococcal sore throat" and was confined to bed for two weeks. During this period she noticed an intermittent, dull ache in the left hip, present usually at night. After getting up, the pattern of symptom-free days and pain at night persisted. The pain was relieved by aspirin. The sedimentation rate was at first normal and later found to be elevated.

Physical examination demonstrated no abnormalities except for tenderness over the left femur anteriorly at the level of the lesser trochanter. There was no limitation of hip motion.

The temperature, pulse and respirations were normal. The blood pressure was 105 systolic, 70 diastolic.

Examination of the blood disclosed a white-cell count of 10,800, with 68 per cent neutrophils, and a hemoglobin of 13.8 gm. per 100 cc. The urine was normal. The erythrocyte sedimentation rate was 30 mm. in one hour. The blood calcium was 9.1 mg., the phosphorus 3.9 mg. and the alkaline phosphatase 3.1 units per 100 cc. A blood Hinton test was negative.

X-ray study revealed a dense area of periosteal new-bone formation associated with the medial anterior aspect of the proximal femoral shaft (Fig. 1). This periosteal new bone was in part differentiated from the course of the bone. In the lateral view a circular zone of rarefaction was observed in the cortex at about the midportion of the area of new-bone formation.

The patient received aspirin on several nights for relief of pain, and was discharged ten days after admission.

Second admission (eight months later). In the interval she continued to go to school. The pain persisted in the medial aspect of the upper third of the left thigh, more constant at rest and disappearing on activity. It recurred regularly at 2 a.m. and was entirely relieved by aspirin. She took up to 8 aspirin tablets daily. There was no recorded temperature elevation at home.

On physical examination the findings were unchanged from those on the previous admission. In the left thigh, about 9 cm. below the anterior superior spine and 1.2 cm. medially, was an area of deep tenderness, measuring 5 by 5 cm. No mass was palpable. The range of motion of the joint was full. There was 1.2 cm. of measurable atrophy of the left thigh, 15 cm. above the patella.

An operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. ERNEST M. DALAND: This was a low-grade type of pain. Apparently it had been that for a number of months. We are told that it started eighteen months previously and that ten months before admission the patient had a streptococcal infection. We know nothing about what happened at the onset of this disease eighteen months before admission, but at one time apparently she did have an infection that laid her up in bed for two weeks. The sedimentation rate was found to be normal, and as time went on it was elevated. She had definite tenderness over the upper femur. The temperature was normal when recorded. The white-cell count

was slightly elevated, and the hemoglobin was essentially normal. The sedimentation rate was elevated and indicated that infection was present. The calcium and phosphorus were normal, as was the phosphatase. May we see the x-ray films?

Dr. Stanley M. Wyman: The lesion, I think, is readily seen on this view of the pelvis, which shows both upper femurs. The left femoral shaft, just below the lesser trochanter, shows an area of marked cortical, possibly some periosteal, thickening with a focus, perhaps 1.5 cm., of sharply defined rarefaction (Fig. 1). This is better seen on slightly more exposed films and is shown in both projections. This view shows what was called a periosteal calcification, apparently separated from the tendon, at least, in part. There is no definite evidence from these films of any focus of increased density within the ovoid rarefaction. There is no evidence of ray formation and no evidence of a soft-tissue mass.

Dr. Daland: The severity of the symptoms was not very great apparently at this time; and after staying in the hospital for ten days and carried only on aspirin, she was sent home. It must have been believed that there was nothing suggesting a tumor at this time, or I think she would not have been allowed to go home. In looking at these films we see a heaping up of calcification by periosteal replacement of bone, which does not look like tumor formation. One would think that if a Ewing tumor were present, we might get a heaping up here of the so-called onion-skin appearance. We would also have a soft-tissue mass, and the appearance of the bone would be unlike this. We would not expect to find a hole in the bone.

She was sent home and returned eight months later. In the interval she continued to go to school, so that the pain could not have been very severe. The pain persisted in the medial aspect of the upper third of the thigh and was more constant at rest, disappearing on activity. The symptoms recurred regularly at 2 a.m. and were entirely relieved by aspirin. This type of pain at night is rather typical of bone pain. We find it in joint disease as well. The classic story of the child with a tuberculous hip or knee is that of a patient who is fairly comfortable in the daytime but has pain at night. In this case there is nothing else to suggest a joint condition. At night there is complete relaxation of muscles, but the patient moves in bed and awakens with severe spasm that brings on pain.

We have, then, a girl with a low-grade condition, pain over a period of eighteen months plus eight months, a little over two years. During that time she was able to carry on and go to school, and the pain did not increase a great deal. Have we the films of the last admission?

Dr. Wyman: We have only the films of the first admission. The others have disappeared, unfortunately. There was essentially no change, however.

Dr. Daland: The duration and type of pain pretty much rule out neoplastic disease in the early examinations. We know that she had an elevated sedimentation rate. It was indicated at one time that she had a considerable degree of infection somewhere. The normal calcium, phosphorus and phosphatase help rule out any type of cystic disease from hyperparathyroidism. The appearance of this lesion was not that of a solitary bone cyst. That is usually a central, medullary condition running down the canal and not showing an overgrowth

Figure 1.

of bone like this, unless there has been a fracture. We do have evidence of a good deal of periosteal proliferation and overgrowth, but more important is the area that was hollow in the center of the reactive bone. This is the sort of thing that one gets in a bone abscess or Brodie's abscess. In Brodie's abscess the pain is low grade and may continue for a number of months without being severe enough to call for any operative treatment. It is rather unusual to find it in the femur. It is much more common in the tibia but is occasionally found in the fibula. This is the age at which one is pretty apt to find a Brodie abscess: anywhere between twelve and twenty — it is rarely seen in adults. I think we can

rule out any type of tumor here. I am pretty certain it is infection. At some time she may have had a low-grade osteomyelitis, and this may have been a residuum of it. The other thing that she might have had was a subperiosteal hematoma, the result of some trauma, although we have no note of such trauma. That would give a heaping up of periosteum like this, but probably would not cause the central hollow in the bone. My opinion is that this was a Brodie abscess.

DR. WYMAN: Would Dr. Daland consider the possibility of osteoid osteoma, whatever that is?

DR. DALAND: Yes.

DR. ROBERT S. PALMER: What is it?

DR. WYMAN: I do not know.

DR. JACOB LERMAN: Were the indications for operation at the second admission any greater than those at the first admission?

DR. TRACY B. MALLORY: No, except that she had not improved, and it was obvious that the lesion would not clear up of its own accord.

CLINICAL DIAGNOSIS

Osteoid osteoma.

DR. DALAND'S DIAGNOSIS

Brodie's abscess of femur.

ANATOMICAL DIAGNOSIS

Osteoid osteoma of femur.

PATHOLOGICAL DISCUSSION

DR. MALLORY: This lesion was explored as is so generally necessary in establishing a diagnosis of a bone lesion. The area of thickening of the periosteum just below the lesser trochanter was found. The cortical bone underlying this area of periosteum was chiseled out, and in the center of the cortical bone a little focus was found, which is shown in this picture (Fig. 2). On the right is the external surface of the cortex, showing irregular thickening beneath the area of periosteal proliferation. In the picture on the left we see the cavity lying within the excised piece of cortical bone. This cavity was filled with a tumor-like mass of material that was rather firm, although distinctly softer than the surrounding cortical bone. Microscopically it was typical of so-called osteoid osteoma. It consisted of interlacing trabeculae of imperfectly calcified osteoid material, and there was the usual increased density of the surrounding cortical bone. Exactly what this lesion was is doubtful as may have been guessed from the questions already asked. Jaffe,* who first described the lesion in 1935, considers it to be a neoplasm of benign character; an unusual feature for neoplasm, however, is that these tumors appear to be entirely self-limited as to growth. In spite of the fact that this had been present two years and under observation many months between the first and second examinations, there was no increase in size during that time. That is characteristic of similar lesions kept under observation for even longer periods. So, if it was a neoplasm, it was one that is incapable of growth beyond a certain very definite and relatively small limit of size. They always lie in the cortical bone. In this case a narrow, recognizable shell of cortical bone separated the lesion from the medullary cavity, and it was not necessary to enter the medullary cavity to remove the lesion. Very persistent pain is extremely characteristic of all examples of the lesion. Sometimes the pain is more severe than that in this case and may be resistant to much stronger therapy than aspirin.

*Jaffe, H. L. Osteoid-osteoma: benign osteoblastic tumor composed of osteoid and atypical bone. *Arch. Surg.* 31:709-728, 1935.

FIGURE 2.

CASE 35182

PRESENTATION OF CASE

A forty-eight-year-old married woman was admitted to the hospital because of pain in the right eye and fever.

Ten days prior to admission the patient began to complain of low-back pain. Two days after this, the pain having subsided, she became nauseated and vomited while riding in a train. On the following day, seven days before admission, she complained again of low-back pain and was found to have a temperature of 104°F. She refused to see a doctor, though she did take three penicillin tablets, which had been left for another patient. The fever allegedly subsided but returned. She continued to have a high temperature and low-back pain until three days before entry, when the right eye became inflamed. Two days before admission, despite her objections, a physician was called. He found her to be quite ill, tested some urine and found it to contain sugar and acetone. She refused hospitaliza-

tion and was given oral fluids and insulin. On the day before entry another physician aspirated some material from the right eye.

For approximately sixteen years the patient had mild polyuria and polydipsia but refused to see a physician. For five to ten years she had occasional ulcers on the lower legs that occurred on the slightest trauma and were very slow to heal. In the two years before admission she had intermittent pain and injection of the right eye, with decreased vision and marked photophobia.

The patient's mother had diabetes for many years. Her father and two brothers died of carcinoma. One sister had a "lump" removed from a breast. The patient's husband was in a tuberculosis sanatorium fifteen and again eight years previously, each time for approximately a year.

Physical examination revealed an obese, critically ill woman. The right conjunctiva was injected, the globe was prominent, and the eye was very tender. The skin was hot and dry. There were a few necrotic lesions on the toes that looked like small areas of gangrene underlying old calluses. Examination of the chest was unsatisfactory owing to poor cooperation, but nothing significant was found. The heart was not enlarged, and a Grade II to III, harsh, high-pitched systolic murmur was heard over the entire precordium, loudest at the apex and toward the sternum. No diastolic murmur was heard. The sounds were of good quality. The abdomen was huge, with a thick panniculus. The dorsalis pedis and the posterior tibial arterial pulsations were not felt. There were scarred and pigmented areas over the lower extremities.

The temperature was 103.6°F., and the pulse 100. The blood pressure was 140 systolic, 70 diastolic.

The urine had a specific gravity of 1.026 and gave a + test for albumin. The white-cell count was 24,900, with 91 per cent neutrophils. The hemoglobin was 16 gm. The blood sugar was 279 mg., and the nonprotein nitrogen 39 mg. per 100 cc., and the carbon dioxide 27 milliequiv. per liter. An x-ray film of the chest showed increased vascular markings throughout both lung fields. At the right apex there was a rounded area of increased density. The aorta was somewhat tortuous. The heart appeared to be somewhat enlarged, with left ventricular prominence. The spinal fluid contained 5 lymphocytes and 210 red cells per cubic millimeter. The protein was 45 mg. per 100 cc. The gold-sol curve showed elevation in the midzone to a maximum of 2. A blood culture taken on admission grew out beta-hemolytic streptococci in both flasks.

The patient received 1,500,000 units of penicillin a day. On the first day of hospitalization she received a total of 90 units of insulin. The temperature dropped to normal, but the fasting blood sugar remained elevated to 346 mg. per 100 cc. The chemosis in the right eye increased, and the process was thought to be a hemorrhagic glaucoma. The ocular tension was 37 as compared to 20 in the left eye. She had immature cataracts in the left eye. She continued to have a fever of 100°F. to 101°F. and a white-cell count varying from 18,000 to a maximum of 27,000. The sensorium cleared somewhat. She received, in addition to the regular injection of penicillin, 25,000 units of penicillin subconjunctivally. There was questionable change in the quality of the heart sounds. On the seventh hospital day it was noted that she was icteric. A Grade I, apical, high-pitched systolic murmur was present, together with a short, high-pitched, coarse systolic sound, best heard in the third left intercostal space but audible at the apex. This second sound was faint and followed shortly by another coarse, high-pitched sound similar to the one following the first sound. There was a questionable, low-pitched, mid-diastolic murmur heard at the apex. There was no edema, rash or petechiae, and the spleen could not be palpated. An electrocardiogram showed partial auriculoventricular block, with a PR interval of 0.25 second. The ST segment was slightly elevated in Leads 1, 2 and 3 and V_4, and the voltage was slightly low. On the eleventh hospital day auricular fibrillation was noted. There was a change in the quality of the heart murmurs, a definite, low-pitched, rumbling diastolic murmur now being present. The heart rhythm later became more regular, with occasional ectopic beats. The temperature remained nearly normal, though the white-cell count was very much elevated. The icterus became more evident. On the twelfth hospital day she lapsed into coma; breathing became shallow, the blood pressure was unobtainable, and she died.

Differential Diagnosis

Dr. W. Wilson Schier: The first significant statement in the history is that this patient was admitted to the hospital because of a fever. Presumably we are dealing with an infectious process from this point on. The low-back pain is mentioned frequently as a cardinal symptom, but my opinion is that this was associated with septicemia. She was acutely ill for ten days preceding hospital admission, and two days before admission she was found to have sugar and acetone in the urine, for which she was given fluids by mouth and injections of insulin.

From the data given I think we are justified in making a diagnosis of diabetes mellitus. This metabolic defect led to a complicated series of events. She apparently suffered from occasional ulcers on the lower legs, which were a product of poor circulation. It was noted that arterial pulsations in the posterior tibial and dorsalis pedis arteries could not be felt — consistent with atheromatous degeneration. The character of the walls of the vessels was not commented on. These areas of ulcera-

tion were a likely portal of entry for the subsequent infection. Buerger's disease is described as occurring rarely in women, and its first sign may be migrating phlebitis. It is much more reasonable to assume that the defective circulation was associated with this patient's diabetic state. She had three penicillin tablets preceding admission, and the remote possibility that a refractory organism resulted from this is only mentioned to condemn inadequate medication. She had gangrene of the toes, which is a common complication of untreated mild diabetes.

Is there anything in the record indicating the nature of the aspirated fluid from the right eye?

Dr. James H. Means: I do not know. She was sent to the Eye and Ear Infirmary.

Dr. Schier: I do not know what to make of the eye lesion — I shall accept the diagnosis of hemorrhagic glaucoma. In the absence of other embolic processes it seems barely possible that she had a mycotic aneurysm of the optic artery to account for her symptoms in the right eye. She had had symptoms in the eye, however, during the two years preceding admission to the hospital, which would be consistent with glaucoma.

I now come to the portion of the discussion that offers richer speculative material. The heart initially was described as not being enlarged on physical examination, but a harsh high-pitched systolic murmur was heard over the entire precordium, loudest at the apex and at the sternum. My inclination is to explain the murmur by dilatation of the left ventricle, although old rheumatic fever cannot be dismissed. In addition to the murmur she had a high temperature, and a blood culture grew beta-hemolytic streptococci. The infection was not overwhelming enough to depress the bone-marrow function, for the white-cell count initially was 24,900, with 91 per cent neutrophils. The heart is described on x-ray examination as being enlarged — may we see the x-ray films?

Dr. Stanley M. Wyman: This film was taken with the patient supine and with the x-ray tube quite close to her; therefore, the apparent enlargement of the heart is not valid because of magnification and because of the relatively high position of the diaphragm. Also, the chest is rotated on the film, so that I think it is not safe to make any conclusion at all about the heart size from this examination. There is probably some calcification in the aortic arch seen at that point. The mention of density at the right apex I am not able to confirm, and I am not impressed by the prominence of the hilar pulmonary vascular shadows in this type of film.

Dr. Schier: You do not see any calcification in the region of the aortic and mitral valves?

Dr. Wyman: I do not believe that calcification in that area would show in this type of film, and she was not fluoroscoped. We do have films of the skull that are very fragmentary because of her condition, I assume, but there is a suggestion on two of these films of a calcified vessel seen through the right orbit and lying in the expected location of the internal carotid artery. This may represent a calcified vessel, but I am unable to be certain of the observation from these poor films.

Dr. Schier: That knocks the props from explaining the murmur by left ventricular dilatation. I suppose arteriosclerosis in the valves would explain the murmur.

The lesion at the right apex might well have been tuberculous — in the welter of symptoms and signs a sputum examination may have been overlooked, and we know that tuberculosis is four or five times more common in diabetic patients than in the population at large.

The patient received massive doses of penicillin, which brought her temperature down only temporarily. I assume that the spinal tap was done to rule out possible meningitis. Was the globulin or the gold-sol curve elevated? What did the culture show?

Dr. Tracy B. Mallory: No globulin is reported.

Dr. James Wyngaarden: The culture was negative.

Dr. Schier: No further mention is made of blood cultures. On the seventh day there was a change in the quality of the heart sounds. First the development of an aortic systolic murmur, a faint second sound and an aortic diastolic murmur. These events and the evidence of septicemia lead me to conclude that this patient developed an acute endocarditis of the aortic valves. The mid-diastolic murmur at the apex became definite four days later and may have been the result of involvement of the mitral valves by the same process or an Austin–Flint murmur associated with the aortic-valve incompetence — resulting from vegetations or perhaps actual perforation of the valves by the destructive process. Why was the heart involved

at all? Old rheumatic heart disease need not be postulated, and the only evidence for a congenital lesion is the description of the murmur heard on admission, which could be consistent with an interventricular septal defect. Were there any evidences of embolic phenomena occurring in the kidney — red cells in particular? The urine is mentioned as showing only albumin. Both focal and diffuse nephritis may be associated with acute endocarditis.

Dr. Mallory: No red cells were observed in four specimens examined.

Dr. Schier: I wonder if anyone would discuss the electrocardiographic changes?

Dr. John W. Hurst: The electrocardiogram showed a partial heart block; the PR interval was 0.27 second, the ST segment was slightly elevated in Leads 1, 2, 3 and V_4 with low voltage. I reported that as consistent with myocarditis if pericarditis could be ruled out. It looked more like myocarditis than coronary-artery disease.

Dr. Schier: The electrocardiographic changes are consistent both with severe infection and coronary-artery disease or rheumatic fever. The changing rhythm might occur with all of these, particularly the last two. With coronary-artery disease I would expect the ST segments to be depressed. Pericarditis associated with endocarditis and myocarditis would be more apt to cause elevation of the ST segments.

It is said that the patient died in icterus, although none was noted on admission. This seems most likely to have been a result of overwhelming sepsis of the liver from infected emboli. Without further clinical and laboratory evidence I cannot rule out cholelithiasis, pancreatitis and hepatitis. There is no mention of cardiac failure, which could cause jaundice, or evidence of large free emboli. Hemachromatosis is unlikely without the finding of pigmentation. Liver disease usually precedes diabetes by months, and I mention it only in passing.

In summary, my diagnoses are diabetes mellitus, acute endocarditis involving the aortic and mitral valves, septicemia, atheromatous arteriosclerosis, hemorrhagic glaucoma of the right eye and possible cardiac enlargement, with terminal hepatitis.

Dr. Richard Clark: What type of endocarditis? Do you mean acute bacterial endocarditis?

Dr. Schier: Yes, acute bacterial — I cannot rule out rheumatic.

Dr. Clark: Would the appearance of the murmurs, in addition to the prolonged PR interval indicating myocarditis together with the ST segments and sounds suggestive of pericarditis as well, fit better with activation of an acute rheumatic process, along with bacterial endocarditis? It is not infrequently seen in combination.

Dr. Schier: I do not know.

Dr. Hurst: One point that might be brought up concerns the diastolic murmur, which you thought might be aortic. Perhaps you were misled by the description in the protocol. That murmur was not of the high-pitched type heard in aortic regurgitation; it sounded more like the scratching of pericarditis. The really changing murmur was at the mitral area rather than the aortic.

Dr. Schier: That would change my opinion of the aortic lesion and would be more consistent with a mitral valvulitis.

Dr. Means: I think that Dr. Schier has covered nearly everything and I believe our line of thought ran similarly to his. I think he quite properly stressed the changing character of the auscultatory findings in the heart. They changed during observation quite strikingly, and there was one period, I am not sure it got into the record, when there was a most amazing phenomenon. There was only one heart sound, a loud clear sound and nothing else, and that, as well as the septic fever, made us all think that something fairly active must be going on in the heart. We considered the possibility of some kind of pyemic process with pus formation in various places. I do not want to say too much about that because I am vague about our ideas, whether they were before autopsy or after autopsy, but we were concerned about why penicillin did not have more effect. We wondered if it was because she had pus that it could not reach.

This woman was an interesting one from the point of view of personality. She had a strong anti-doctor complex, and we thought that a good deal of her pitiable condition might have been prevented if she had consulted a doctor twenty years previously. The obesity was tremendous and had a good deal to do with the diabetes. She neglected the infections and so forth.

Dr. Alfred Kranes: Did the blood cultures become negative after treatment started?

Dr. Means: I cannot remember; I think they did

DR. MALLORY: Only that single culture is reported.

CLINICAL DIAGNOSES

Acute bacterial endocarditis.
Cholemia.
Diabetes mellitus.

DR. SCHIER'S DIAGNOSES

Diabetes mellitus.
Acute endocarditis, mitral.
Septicemia.
Atheromatous arteriosclerosis.
Hemorrhagic glaucoma of right eye.
Terminal hepatitis.

ANATOMICAL DIAGNOSES

Endocarditis, acute bacterial, of mitral valve.
Abscesses, multiple, of heart, spleen and kidneys.
Portal cirrhosis of liver, post-necrotic type.
Pulmonary tuberculosis, active, right apex.
Patent ductus arteriosus.
(Diabetes mellitus.)
Arteriosclerosis, generalized, moderate.

PATHOLOGICAL DISCUSSION

DR. MALLORY: Autopsy confirmed the diagnosis of bacterial endocarditis. When the pericardium was first opened we found a small amount of fresh blood but no inflammatory changes to indicate an infectious pericarditis. On inspecting the surfaces of the heart, we noted several small superficial abscesses, one of which immediately adjoined a coronary vessel; the surrounding tissues were ecchymotic, and we thought there had been a little leakage of blood from the coronary artery to account for the blood in the pericardium. The mitral valve showed a very large shaggy vegetation extending almost two thirds around the orifice. The chordae tendineae were nearly normal — possibly slightly thickened. The annulus of the mitral valve showed calcification, and there probably was a slight underlying old rheumatic heart disease. The ductus arteriosus was found to be still patent. At the pulmonary end it measured nearly 1 cm. in diameter but as it approached the aortic orifice it narrowed markedly and the opening into the aorta measured only 1 mm. in diameter. I do not believe a significant amount of blood was passing through and doubt that any of the murmurs were explained on that basis; they probably all arose from extensive mitral involvement. Both infarcts and pyemic abscesses were found in various organs. In the spleen there was one bland infarct and one septic one. There were multiple abscesses in the kidney but no characteristic infarcts. The liver showed a severe degree of cirrhosis of the coarsely nodular, post-necrotic type, and there were demonstrable varices in the lower portion of the esophagus. We found no gross abscesses in the liver, but, on microscopical examination, numerous foci of sepsis were found and I think the combination of old liver disease and scattered fresh necrosis constituted adequate explanation of the terminal liver insufficiency. One large, well encapsulated tuberculous focus was present at the apex of the right lung.

We did not have permission to examine the head.

DR. KRANES: Did the post-mortem cultures grow the same organisms that were found during life?

DR. MALLORY: We grew a pure culture of beta-hemolytic streptococci from one of the heart abscesses. Cultures from the kidney grew mixed organisms, both beta-hemolytic and alpha-hemolytic streptococci and colon bacilli.

DR. MEANS: Could penicillin have been responsible for the colon bacilli? Do they not come in when penicillin is administered?

DR. MALLORY: They do in open lesions such as bronchiectatic cavities but I do not believe they do within the tissues of the body.

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THE HEALTH PROTECTION CLINIC

Different projects have been underway for varying lengths of time whereby the person interested in his health problems may undergo a "screening" technic to eliminate, so far as possible, the presence of certain diseases. It seems logical that proposals should be made to amalgamate these screening processes — for cancer, for heart disease, for diabetes, for tuberculosis and for other diseases — under one roof and at one time, for what they are worth.

It is difficult to evaluate this worth on a statistical basis. The most objective mathematician would hardly dare to set an exact price on the saving of a life, and screening technics are capable not only of discovering an uncertain proportion of persons with incipient disease out of the total number examined, but of assuring many of the others of their general good health at the time of examination.

The screening technic itself must naturally not be overrated nor the object of too much enthusiasm. The course of disease may be inexorably progressive or it may first become discoverable after the technic of discovery has declared the patient to be apparently physically fit. Nevertheless, much more can be done to eradicate incipient disease or to mitigate its course than was possible a decade ago.

An objective to be achieved if the health protection clinic is to serve its purpose is that of streamlining its technic. If the processes of discovering the presence or absence of disease are to be made possible on a large scale, they must be so simple that the whole plan does not fall from the sheer weight of man hours involved.

If health protection services, moreover, are to be offered freely or in any other manner to the public, social and economic problems must be settled. Whether the means test, as either an indignity or a dignity, is to be employed must be decided. The method of reference of patients to the clinics and the disposition of them after screening must be determined, as well as the financing of the whole enterprise. The interests of the private practitioner must be protected at all costs, for he is still and will remain the most effective and the most highly considered dispenser of health services.

In this respect it may be pertinent to observe that improvements in health services, as they have generally been offered, have seemed usually to redound to the advantage of the practitioner. Obviously the health protection clinic as a public service is social medicine. Whether it represents also socialized medicine is a matter of definition, but if it does, the country has enjoyed or suffered under socialized medicine to some degree and in various forms for a number of years.

The important point is that if the practice of medicine continues to change its form, as it has been doing since the Babylonian physicians first added liver divination to their prognostic armamentarium, its course will be truer and its benefits greater if the medical profession goes with it and guides it, and

does not try simply to interpose inadequate obstacles in its path.

It seems reasonable that pilot studies at least should be made in the establishment of health protection clinics. From the lessons that they teach new bearings can be taken to guide progressive medicine on its course.

GROUP HEALTH ASSOCIATION

TEN years ago in Washington, D. C., bitter controversy arose regarding the so-called Group Health Association, and much opposition to its activities was encountered. Time has passed and views have changed, and elsewhere in this issue of the *Journal* a report appears of the first ten years of that organization, about whose activities there was such a wide difference of opinion at that time regarding its ethical and legal status.

Statistics may be a notoriously unsound basis for the drawing of conclusions, and it is important to note that the author himself states that at least one reason for the fact that the mortality rates compared very favorably with those from the community at large may be that the group so covered was in a higher financial bracket than some other groups. However, the average family income of the group studied would hardly, in these days, be considered much above the lines drawn by various insurance companies to separate those in low income brackets from those in the medically indigent class.

One of the criticisms often made of the attitude of the medical profession is that it has been slow to do anything in the way of constructive planning to meet the needs of present conditions. It is with this criticism in mind that the *Journal* is publishing the article in question, which deals with the experience of a small group in a specific area in the country. It is important to appreciate that it is the *local* physicians who should be in control of the *local* needs of any community, whatever the means or agency may be that attempts to provide good medicine at a cost within the range that the individual can pay. If state and federal aid is needed — and it seems likely that it will be — such aid must not predicate jurisdiction over the *local* needs and problems.

How to co-ordinate local, with general over-all supervision is the question that is fundamental to success.

DIHYDROSTREPTOMYCIN

DIHYDROSTREPTOMYCIN, a new derivative of streptomycin, has recently become available. It is made from salts of streptomycin by a process of catalytic hydrogenation that adds about one molecule of hydrogen to each molecule of streptomycin.[1-4] The resulting dihydrostreptomycin salts appear to be more stable than the parent compounds in that, unlike the latter, they are not inactivated by cysteine, hydroxylamine or semicarbazide and are not degraded by heating with alkali. Comparative tests of the activity of dihydrostreptomycin and its parent streptomycin have shown that both have essentially the same activity in vitro against most bacteria.[2, 3, 4-7] Against some species or strains of organisms, including occasional strains of tubercle bacilli and of salmonella, the dihydrostreptomycin, weight for weight, may be only two thirds or even as little as a fourth as active as streptomycin.[4, 8] In no case has a dihydrostreptomycin compound been found to be more active than its parent streptomycin.[5] The antibacterial actions of dihydrostreptomycin and streptomycin are similarly affected by changes in pH[4] and in certain components of the medium.[4, 8] Organisms resistant to streptomycin are equally resistant to dihydrostreptomycin.[4, 6, 7] Some workers have suggested that the antibacterial activity of dihydrostreptomycin results from its being oxidized to streptomycin by bacteria.[9]

In animal experiments[4, 5, 10] and in man[10, 11] both compounds appear to be absorbed and excreted in essentially the same manner. They appear to be about equally active, weight for weight, against infections in animals with strains of tubercle bacilli[5, 6, 7, 12] and with most of the pathogenic organisms that have been tested.[1, 4, 5] The parent streptomycin, however, appears to be more active on a weight basis against some of these infections.[1] Dihydrostreptomycin has no therapeutic effect on infections of animals with organisms that are resistant to streptomycin.[6, 7]

The only therapeutic advantage of dihydrostreptomycin has been in toxicity, although dihydrostrepto-

mycin has produced all the toxic effects observed with streptomycin.[6] In animals it has been found that the acute toxicity — that is, the amount that is fatal for an animal in a single dose — is essentially the same for both compounds.[4, 5] The histamine-like effect obtained from one lot of dihydrostreptomycin was only about a sixth of that produced by the lot of streptomycin from which it was derived.[4] In chronic toxicity experiments dihydrostreptomycin proved to be less toxic, in that larger doses given over a longer period were required to produce neurotoxic effects equivalent to those obtained from streptomycin.[6] There was also some indication that the local ulceration and induration resulting from dihydrostreptomycin in animals were less marked than those produced by streptomycin.

The effects of the use of dihydrostreptomycin in the treatment of human beings have thus far been reported in only a small number of patients, some of whom were treated with impure materials.[10, 13] The results in these cases indicate that dihydrostreptomycin produces all the toxic effects that have been observed from streptomycin. Dihydrostreptomycin, however, may have somewhat less neurotoxicity. The impression gained from the few cases thus far studied after the use of large doses of dihydrostreptomycin is that the vestibular damage occurs later and less uniformly and may be less disabling than that after streptomycin.

The most striking observations, however, have been made in patients who have been unable to tolerate streptomycin because of fever, rashes or other evidences of sensitization. In such patients dihydrostreptomycin has been well tolerated, and it has been possible to continue the treatment of such patients without ill effects.[8, 13] Some of the experiences with early lots suggested that dihydrostreptomycin was more irritating locally, particularly when given intrathecally.[13] By the latter route streptomycin was said to be preferable,[13] although the irritation observed from the intrathecal injections of dihydrostreptomycin may have been the result of the use of impure preparations. Dihydrostreptomycin has shown the same tendency as streptomycin to produce resistant organisms and neither compound is effective in the treatment of infections with streptomycin-resistant organisms.[8]

It therefore appears that dihydrostreptomycin may prove useful in the treatment of patients who show allergic sensitivity reactions to streptomycin. It may also prove to be more desirable than streptomycin in patients who require large doses or prolonged treatment as in the therapy of tuberculosis. Further experience is necessary before even these conclusions can be justified.

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INDIAN GIVER

The Great White Father, despite bumper budgets of recent years, has turned out to be, in the case of his copper-colored children, an Indian giver. The original possessor of the country's fields and forests, now one of the world's most forgotten displaced persons, surrendered these possessions (after a struggle) for the opportunity of living in squalor as a permanent Guest of the Nation.

In both 1947 and 1948, according to *Washington Report on the Medical Sciences*, teams selected by the American Medical Association and sponsored by the Department of the Interior inspected Indian reservations in the West and found appalling conditions of health and living. The Bureau of Indian Affairs of the Department of the Interior then asked for a "minimum needs" budget of $9,163,814, from which the Budget Bureau promptly slashed $1,500,000.

Medical Director Fred Foard, protesting both the budget cut and conditions that make it impossible

to provide the country's quarter of a million reservation Indians with good medical and public-health care, plans to fight for "reforms of a system in which field medical officers are wholly subordinate to lay commissioners, salaries of doctors and nurses are inadequate and central direction is impossible."

These wards of the state are obviously permanently displaced; perhaps a little more of the nation's charity might begin at home.

MASSACHUSETTS MEDICAL SOCIETY

DEATH

HANNIGEN — Robert C. Hannigen, M.D., of Amesbury, died on December 16. He was in his seventy-second year.

Dr. Hannigen received his degree from Medical School of Maine, Portland, in 1901.

His widow, three sons and a daughter survive.

NOTES FROM THE MEDICAL EXAMINER

ACID PHOSPHATASE TESTS AS EVIDENCE OF RAPE

The family physician is usually the first person called when aid or advice is sought by the victim of a sexual assault. His duties in such cases include both the care of the patient and the accumulation of evidence likely to be of value in establishing identity or guilt of the assailant. The evidence is twofold: that of physical violence suffered by the victim, as shown by bruises, scratches, or other injuries of her body, and evidence of ejaculate on or about her body. Since rape of a child or woman not under the influence of drugs or submitting from fright is practically impossible without leaving marks of violence on the extremities or genitalia, the absence of such injuries casts serious doubts on the fact of the alleged rape even though semen may be demonstrated.

The identification of spermatozoa in stained smears from the victim's perineum or vagina is obviously the best proof of the presence of semen. Identification is usually made on material from four sources: vaginal content, water suspensions of material clinging to the perineum or thighs, soiled pubic hair and stained clothing. In the first two instances smears should be made and dried promptly. The specimens should be submitted in dust-protective wrapping to a laboratory for staining and study by an expert.

Direct demonstration of spermatozoa is frequently impossible, owing either to destruction of the cells in the vagina or their absence in the ejaculate (aspermia or vasectomy). The survival of spermatozoa in the vagina after coitus varies from thirty minutes to twenty-four hours.[1] The phosphatase test, because it does not depend on intact sperm cells, is a useful adjunct in the study of material suspected of representing ejaculate. The specimens to be collected are clothing bearing stains possibly due to semen, and if feasible, aspirated fluid vaginal content. Stains should be dried; fluid should be refrigerated in a sterile container until testing is done.

Determination of acid phosphatase as a test for seminal stains on clothing was first described by Lundquist and Rasmussen in 1945, and extensive studies by Riisfeldt[2] and Hansen appeared in 1946. The procedure is based on the fact that ejaculate contains a large proportion of prostatic fluid and therefore a high concentration of acid phosphatase derived from prostatic glandular secretion. The demonstration of this enzyme consists of several steps:

Preparation of a saline extract of the dried stain or dilutions of the vaginal content. In stained cloth, the areas to be tested may be selected under ultraviolet illumination, which causes semen as well as many other biologic fluids to fluoresce.

Determination of the acid phosphatase activity of the saline extract by measurement of the phenol released by an aliquot of the extract from a phenylphosphate substrate in one hour under controlled conditions.[3]

Calculation of the acid phosphatase activity of the original specimen in units per cubic centimeter. This is accomplished directly with liquid specimens and in the case of dried stains by application of a factor that relates area of dried stain to cubic centimeter of original fluid causing that stain. The latter procedure is derived from experiments in similar samples of cloth using milk as the fluid, since the spreading power of milk is similar to that of semen.[4] One unit of acid phosphatase activity is that of 1 cc. of original fluid that will hydrolyze 1 mg. of phenol in one hour at pH 4.9 and 37° F.

The acid phosphatase content of all body fluids other than prostatic secretion is less than 20 units, whereas that of ejaculate varies from 400 to 8000 units per cubic centimeter and is high even in the presence of azoospermia or aspermia.[2] Dried stains or fluid found to contain more than 100 units per cubic centimeter of original fluid are interpreted as indicative of the presence of semen in the specimen.

The technic has gained favor in medicolegal investigation, since it allows the recognition of semen long after the ejaculation or assault has occurred. Riisfeldt demonstrated increased phosphatase activity corresponding to the content of semen in vaginal fluid six and twelve hours after coitus but not after twenty-four hours. He

found that the admixture of semen with urine, blood, urethral secretion, sputum, sweat, lachrymal fluid, milk, tea, coffee and cocoa had no deleterious effect upon the phosphatase activity. Likewise, penicillin or sulfathiazole did not affect the phosphatase activity. Dried stains retained activity for several months.

Evidence of the presence of semen on a garment in a case of rape, obtained by the use of the acid phosphatase test, has so far been introduced in the Superior Court in Massachusetts on only one occasion. There is, however, little reason to doubt that the test will be used frequently, and no difficulty is anticipated in having such evidence accepted by the Court, provided it is introduced by a qualified expert.

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Russell S. Fisher, M.D.

Research Fellow, Department of Legal Medicine

Harvard Medical School

MISCELLANY

RICHARDSON LECTURES

The Massachusetts General Hospital on April 8 inaugurated the Edward Peirson Richardson Lectures in Medical Science. A portrait of Dr. Richardson, former chief of the surgical services, was unveiled, and the first lecture in the series was delivered by R. S. Pilcher, M.S., F.R.C.S., professor of surgery at University College Hospital, London, on Bronchiectasis.

CORRESPONDENCE

LOCAL REGISTRATION OF PHYSICIANS

To the Editor: The Franklin District Medical Society has recently discovered that approximately 40 per cent of the physicians living and practicing in Greenfield have not been registered with the town clerk as required under the provisions of Chapter 55, Section 3, of the Acts of 1917. This deficiency has been corrected, but it brings up the question whether other fellows of the Massachusetts Medical Society have failed to register with their town or city clerks.

It is important that correct registration be completed, because the physician is liable to a fine up to $100.00 for not so registering; the town or city clerk is liable to a fine up to $15.00 for each physician not registered by him; and, I am informed, should a physician in this status be involved in court procedures, should be adequate in all other respects, and should even successfully defend himself in a malpractice suit he would not be entitled to the protection of the law because he was practicing medicine illegally.

Lawrence R. Dame, M.D.

Greenfield, Massachusetts

"SCIENCE AND IMMORTALITY"

To the Editor: It was with great pleasure that I read the editorial "The Second Mile," which, to me, embodies the philosophy of the patient-doctor relationship.

Medical school seems to be a place where many do not "have the supporting strength of a belief in the hereafter" and a place where editorials such as yours should do an immeasurable amount of good.

Being a New Hampshire "Yankee" myself, I feel doubly proud to know that a New England group has sponsored the publication of thoughts such as these, which are so greatly needed.

Edward H. Robinson, 1949

Jefferson Medical College

BOOK REVIEWS

Medullary Nailing of Küntscher. By Lorenz Böhler, M.D. First English edition, revised by the author. Translated from the eleventh German edition by Hans Tretter, M.D. 4°, cloth, 386 pp., with 1261 illustrations. Baltimore: Williams and Wilkins Company, 1948. $7.00.

This is the most authoritative study on the medullary nailing of Küntscher. Professor Böhler has had a most extensive experience in the treatment of fractures by the older conventional means. This should make his judgment of the new technic particularly valuable. The discussion of indications, complications and end results is based upon a review of 600 patients with fractures treated by medullary nailing. The author considers closed medullary nailing superior to all previously known methods in fresh fractures of the femoral shaft. But not all fractures are suitable for this method. The general condition of the patient must be good, with healthy condition of the skin. It is easiest to use in fractures of the middle third. In fractures of the tibia and humerus equally good results and no greater difficulty are encountered with the older methods. The indications for intramedullary nailing are not so wide as universally supposed. This method should not be used for the clavicle or for fractures near a joint. It should be employed only by physicians of wide experience in handling fractures. In compound fractures the danger of infection is great. There is no advantage in nailing in the presence of pseudarthrosis. The complications are not often seen, but death (from shock), infection, wrong placement of the nail, splitting of the bone and breaking of the nail occur. There is an excellent chapter on the problem of callus. Callus is inhibited by the presence of the nail. Without proper impaction there may be distraction of the fragments preventing union. This book can be recommended as the best and most authoritative discussion on the intramedullary nailing of Küntscher that has so far been published.

Sterility and Impaired Fertility: Pathogenesis, investigation and treatment. By Cedric Lane-Roberts, C.V.O., M.S., F.R.C.S., F.R.C.O.G., Albert Sharman, M.D., Ph.D., M.R.C.O.G., Kenneth Walker, M.A., M.B., B.C. (Cantab.), F.R.C.S., F.I.C.S., B. P. Wiesner, D.Sc., Ph.D., F.R.S.E., and Mary Barton, M.B., B.S. 8°, cloth, 400 pp., with 96 illustrations and 18 tables. New York: Paul B. Hoeber, Incorporated, 1948. $6.50.

This book is a second edition, although not so stated on the title page, of a work first published in 1939. This edition has been revised in the light of recent developments, especially in diagnosis and therapy. In diagnosis the precise use and interpretation of postcoital tests and the use of invasion tests are discussed. The technics of semen analysis and of determination of pregnanediol in urine have been simplified. The importance of extended use of cyclical temperature records for the study of ovarian function, anovulatory cycles, inadequate luteinization and the diagnosis of impending abortion is considered. Also, the value of the important tubal insufflation diagnostic test and its validity, and urinary hormone analysis are discussed. In the field of therapy the use of endocrine preparations, the sulfonamides and penicillin, and of x-ray stimulation, especially of the pituitary body, in cases of anovulation, are singled out for special mention. The controversial method of artificial insemination is fully discussed in its clinical, legal, moral and social aspects. The question of terminology is clarified. The text is divided into two parts, the first being devoted to male and the second to female fertility and sterility.

The authors are British, and the printing was done in Great Britain and the book bound in the United States. The publication is excellent in every way. A good legible type is well printed on a coated paper. A number of appendixes, a long bibliography and a good index conclude the volume. The book is recommended for all medical libraries as a standard reference work and to all persons interested in the subject.

OFFICERS OF THE MASSACHUSETTS MEDICAL SOCIETY, 1948–1949

White Star

DR. ARTHUR W. ALLEN, *President-Elect*

PROGRAM OF THE ONE HUNDRED AND SIXTY-EIGHTH ANNIVERSARY OF THE MASSACHUSETTS MEDICAL SOCIETY

Monday, Tuesday, Wednesday and Thursday, May 23, 24, 25 and 26, Worcester Memorial Auditorium and Hotel Sheraton, Worcester

The Registration Desk will be located on the Stage of the Auditorium, and all who attend the meeting are requested to register.

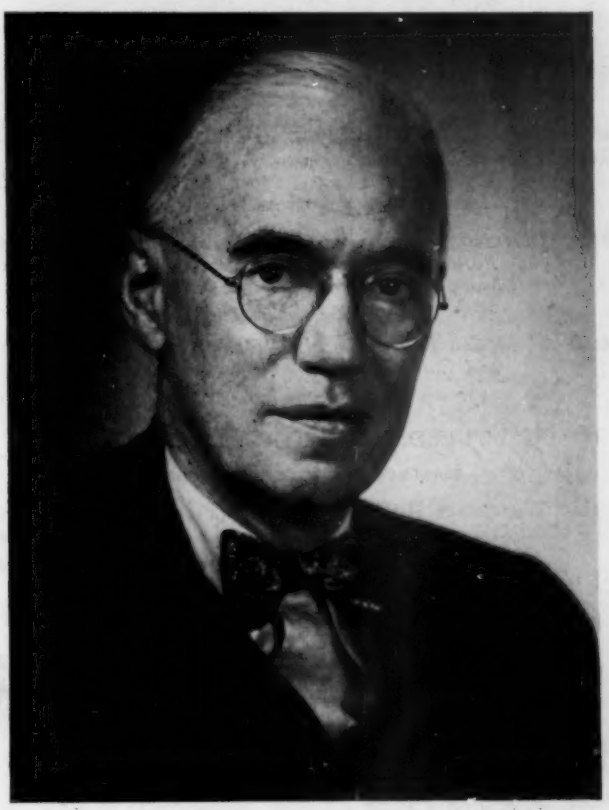
MONDAY AFTERNOON, MAY 23

4:30 **Supervising Censors' Meeting** (Room E, Hotel Sheraton).

6:00 **Cotting Supper for Councilors** (Ballroom, Hotel Sheraton).

Bachrach

Dr. Daniel B. Reardon, *President*



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Dr. Donald Munro, *Vice-President*

MONDAY EVENING, MAY 23

7:00 **Annual Meeting of the Council** (Ballroom, Hotel Sheraton).

TUESDAY MORNING, MAY 24

First General Session

Little Theater, Worcester Memorial Auditorium

Dr. John J. Dumphy, *Chairman*

Dr. Bancroft C. Wheeler, *Co-chairman*

8:55 *A Motion Picture Entitled "Dermatoses of Industrial Workers."*

9:30 *A Public-Health Heart Program — First Report.* Dr. Egon E. Kattwinkel, Newton. Chief of cardiology, Newton-Wellesley Hospital. Drs. Vlado A. Getting, Ernest M. Morris, H. M. Lombard and Lewis C. Robbins.

9:55 *The National Health Service — Recommendations of the Hoover Commission.* Dr. Hugh R. Leavell, Boston. Professor of public-health practice, Harvard School of Public Health.

10:20 *Compulsory Health Insurance. Is this the Best Way to Improve Medical Care?* Dr. Frank H. Lahey, Boston. Director, Lahey Clinic.

11:00 **Annual Meeting of the Massachusetts Medical Society** (Little Theater, Worcester Memorial Auditorium).

Annual Oration (following annual meeting): Some Responsibilities of Medical Education. Dr. C. Sidney Burwell, Brookline. Dean, Harvard Medical School.

Annual Luncheon (following annual oration): tickets must be procured in advance of the meeting.

TUESDAY AFTERNOON, MAY 24

Second General Session

LITTLE THEATER, WORCESTER MEMORIAL AUDITORIUM
DR. EDWARD P. BAGG, *Chairman*
DR. DONALD MUNRO, *Co-chairman*

2:00 *Pitfalls in the Diagnosis of Diarrhea.* DR. HUGH TATLOCK, Northampton. Staff physician, Cooley-Dickinson Hospital; consultant in medicine, Veterans Administration Hospital.

2:15 *Pitfalls in the Diagnosis of Continued Fever.* DR. FRANKLIN K. PADDOCK, Pittsfield. Attending physician, Pittsfield General Hospital.

2:30 *Pitfalls in the Diagnosis of Persistent Cough.* DR. PAUL R. DUFAULT, Rutland. Superintendent, Rutland State Sanatorium.

2:50 *Evaluation of the Papanicolaou Technic in the Diagnosis of Malignant Disease.* DR. HOWARD ULFELDER, Winchester. Clinical associate in surgery, Harvard Medical School; assistant surgeon, Massachusetts General Hospital; senior surgeon, Pondville Hospital.

3:15 *The Present Status of the Management of Thyroid Disease.* DR. GEORGE CRILE, JR., Cleveland, Ohio. Department of Surgery, Cleveland Clinic.

3:40 *Hospital Administration as a Medical Specialty.* DR. T. STEWART HAMILTON, Newton. Director, Newton–Wellesley Hospital.

4:05 *Observations on Reduced Incidence of Embolism: Postoperative ambulation dicumarol routine.* DR. JAMES C. MCCANN, Worcester. Senior surgeon, St. Vincent Hospital.

4:30 *A Motion Picture Entitled "Anomalies of the Bile Duct and Blood Vessels."*

TUESDAY EVENING, MAY 24

BALLROOM, HOTEL SHERATON

8:15 **Worcester Hofbrau Night**. (Free beer, free entertainment and music at the invitation of the Worcester District Medical Society.)

WEDNESDAY MORNING, MAY 25

Third General Session

LITTLE THEATER, WORCESTER MEMORIAL AUDITORIUM

DR. GEORGE R. DUNLOP, *Chairman*

DR. FRED H. ALLEN, *Co-chairman*

8:50 *A Motion Picture Entitled "Management of the Failing Heart."*

Symposium on Gastric and Duodenal Ulcer

9:25 *Medical Aspects.* DR. SARA M. JORDAN, Boston. Director, Department of Gastroenterology, Lahey Clinic; physician, New England Baptist Hospital and New England Deaconess Hospital.

9:50 *The Roentgenologist's Contribution.* DR. JOHN D. CAMP, Rochester, Minnesota. Professor of radiology, Mayo Foundation; consultant in roentgenology, Mayo Clinic.

10:15 *The Management of Hemorrhage.* DR. BURRILL R. CROHN, New York City. Consultant in gastroenterology, Mt. Sinai Hospital; consultant, Veterans Administration, Halloran Hospital, Staten Island, New York.

10:40 *The Surgeon's Point of View.* DR. JOHN D. STEWART, Buffalo, New York. Professor of surgery and chairman of the Department of Surgery, University of Buffalo School of Medicine.

WEDNESDAY MORNING, MAY 25

LITTLE THEATER, WORCESTER MEMORIAL AUDITORIUM

11:05 **The Shattuck Lecture.** La médecine du coeur.

DR. PAUL D. WHITE, Boston. Clinical professor of medicine, Harvard Medical School; consultant in medicine, Massachusetts General Hospital; executive director, National Advisory Heart Council.

WEDNESDAY NOON, MAY 25

Section Meetings and Luncheons

12:00 m. — 2:00 p.m.

Section of Medicine

STAGE, WORCESTER MEMORIAL AUDITORIUM

DR. LAURENCE B. ELLIS, Cambridge, *Chairman*
DR. ALLEN S. JOHNSON, Longmeadow, *Vice-chairman*
DR. JAMES A. HALSTED, Dedham, *Secretary*

Therapeutic Advances in the Treatment of Diarrheal Diseases of Inflammatory Origin. DR. BURRILL R. CROHN, New York City. Consultant in gastroenterology, Mt. Sinai Hospital; consultant, Veterans Administration, Halloran Hospital, Staten Island, New York.

Section of Pediatrics

ASSEMBLY ROOM, WORCESTER MEMORIAL AUDITORIUM

DR. W. BRADFORD ADAMS, Springfield, *Chairman*
DR. GERALD N. HOEFFEL, Boston, *Secretary*

Problems of Pediatric Radiology. DR. EDWARD B. NEUHAUSER, Cambridge. Radiologist, The Children's Hospital, Boston.

Section of Radiology

MALE CHORUS ROOM, WORCESTER MEMORIAL AUDITORIUM

DR. ALBERT M. MOLONEY, Boston, *Chairman*
DR. LAURENCE L. ROBBINS, Boston, *Secretary*

Significant Roentgenologic Findings in Intracranial Disease. DR. JOHN D. CAMP, Rochester, Minnesota. Professor of radiology, Mayo Foundation; consultant in radiology, Mayo Clinic.

Section of Physical Medicine

MUSICIANS' ROOM, WORCESTER MEMORIAL AUDITORIUM

DR. DAVID C. DITMORE, Boston, *Chairman*
DR. ARTHUR L. WATKINS, Boston, *Secretary*

Rehabilitation of the Patient with Hemiplegia. DR. WILLIAM B. SNOW, New York City. Assistant professor of medicine, Columbia University, College of Physicians and Surgeons; director, Department of Physical and Occupational Therapy, Columbia Presbyterian Medical Center.

WEDNESDAY AFTERNOON, MAY 25

Fourth General Session

LITTLE THEATER, WORCESTER MEMORIAL AUDITORIUM

DR. DONALD B. CHEETHAM, *Chairman*
DR. LAWRENCE R. DAME, *Co-chairman*

2:00 *Pitfalls in the Diagnosis of Backache.* DR. CHARLES E. AYERS, Worcester. Orthopedic surgeon, The Memorial Hospital.

2:15 *Pitfalls in the Diagnosis of Rectal Bleeding.* DR. E. PARKER HAYDEN, Boston. Associate visiting surgeon, Massachusetts General Hospital.

2:30 *Pitfalls of Chemotherapy and Antibiotics in Surgery.* Dr. Allen G. Rice, Springfield. Consulting surgeon, Springfield Hospital.

2:50 *Intellectual and Emotional Evaluation of the Sick Child.* Dr. James Marvin Baty, Belmont. Professor of pediatrics, Tufts College Medical School; physician-in-chief, Boston Floating Hospital. Dr. Rowland G. Freeman, Millis. Assistant professor of psychiatry, Tufts College Medical School and psychiatrist, Boston Floating Hospital.

3:15 *Diabetes — Some Fundamental Considerations.* Dr. W. Richard Ohler, Boston. Chief, Diabetic Clinic, Boston City Hospital; president, New England Diabetic Association.

3:40 *The Problem of Diagnosis and Management of Chronic Recurrent Headache.* Dr. H. Houston Merritt, professor of neurology, New York City. Columbia University, College of Physicians and Surgeons; director of service of neurology, Neurological Institute, Presbyterian Hospital.

4:05 *Mechanical and Clinical Considerations in the Medical Care of Patients with Low Back Pain.* Dr. William B. Snow, New York City. Assistant professor of medicine, Columbia University, College of Physicians and Surgeons; director, Department of Physical and Occupational Therapy, Columbia Presbyterian Medical Center.

4:30 *A Motion Picture Entitled "Fractures: An introduction."*

WEDNESDAY EVENING, MAY 25

6:00 **Cocktail Party** (Crystal Room, Hotel Sheraton).

Tickets must be procured in advance.

7:00 **Annual Dinner of the Massachusetts Medical Society** (Ballroom, Hotel Sheraton).

Presiding:

Dr. Daniel B. Reardon, president, Massachusetts Medical Society.

Guest Speaker:

Roscoe Pound, LL.D., University Professor, Emeritus, Harvard University; formerly dean, Harvard Law School. The Professions in the Society of Today.

Ladies may be invited to attend the dinner.

Tickets for the dinner *must* be procured in advance of the meeting.

THURSDAY MORNING, MAY 26

Fifth General Session

Little Theater, Worcester Memorial Auditorium

Dr. James T. Brosnan, *Chairman*

Dr. Norman B. McWilliams, *Co-chairman*

9:00 *A Motion Picture Entitled "Physiology of Normal Menstruation."*

9:25 *Some Problems in Interpretation and Management of Chondromatous Tumors in Bone.* Dr. Granville A. Bennett, Chicago. Professor and head of the Department of Pathology, University of Illinois College of Medicine.

9:50 *Diagnostic Procedures in Virus Diseases.* Dr. F. Sargent Cheever, Wellesley. Assistant professor of bacteriology and immunology, Harvard Medical School.

10:15 *Management of Abnormal Uterine Bleeding.* Dr. George Van S. Smith, Brookline. William H. Baker Professor of Gynecology, Harvard Medical School; surgeon-in-chief, Free Hospital for Women, Brookline.

10:40 *Fetal Loss as Influenced by the Administration of Diethylstilbestrol.* Dr. Olive Watkins Smith, Brookline. Director, Fearing Research Laboratory, Free Hospital for Women, Brookline.

11:05 *Skin Manifestations of Avitaminoses.* Dr. Chester N. Frazier, Boston. Edward Wigglesworth Professor of Dermatology, Harvard Medical School; chief, Dermatological Service, Massachusetts General Hospital.

11:30 *Present Concepts of Nutrition.* Dr. Fredrick J. Stare, Newton. Professor of nutrition, Harvard School of Public Health; associate in medicine, Peter Bent Brigham Hospital.

THURSDAY NOON, MAY 26

Section Meetings and Luncheons

12:00 m. — 2:00 p.m.

Section of Surgery

Stage, Worcester Memorial Auditorium

Dr. Robert E. Gross, Boston, *Chairman*

Dr. Franklin G. Balch, Jr., Boston, *Secretary*

Some Studies in Gastric Secretion of Surgical Interest. Dr. Francis D. Moore, Boston. Moseley Professor of Surgery, Harvard Medical School; surgeon-in-chief, Peter Bent Brigham Hospital.

Section of Obstetrics and Gynecology

Male Chorus Room, Worcester Memorial Auditorium

Dr. James F. Conway, Brookline, *Chairman*

Dr. Daniel J. McSweeney, Boston and Milton, *Vice-chairman*

Dr. Duncan E. Reid, Boston, *Secretary*

Management of Dysfunctional Uterine Bleeding. Dr. George Van S. Smith, Brookline. William H. Baker Professor of Gynecology, Harvard Medical School; surgeon-in-chief, Free Hospital for Women, Brookline.

Section of Dermatology and Syphilology

Female Chorus Room, Worcester Memorial Auditorium

Dr. Francis P. McCarthy, Boston, *Chairman*

Dr. Alfred Hollander, Springfield, *Secretary*

Quantitative Serologic Test for Syphilis. Dr. Chester N. Frazier, Boston. Edward Wigglesworth Professor of Dermatology, Harvard Medical School; chief, Dermatological Service, Massachusetts General Hospital.

Section of Anesthesiology

Assembly Room, Worcester Memorial Auditorium

Dr. Morris J. Nicholson, Boston, *Chairman*

Dr. Jacob Fine, Beverly, *Secretary*

A New Plan for Graduate Teaching of Anesthesiology. Dr. Robert D. Dripps, Philadelphia. Professor of anesthesiology, University of Pennsylvania School of Medicine.

Section of Physiology and Pathology

Musicians' Room, Worcester Memorial Auditorium

Dr. Monroe J. Schlesinger, *Chairman*

Dr. Donald A. Nickerson, *Secretary*

Diagnosis of Certain Skeletal Disorders That Give Rise to Cystic Lesions in Roentgenograms. Dr. Granville A. Bennett, Chicago. Professor and head of the Department of Pathology, University of Illinois College of Medicine.

THURSDAY AFTERNOON, MAY 26

Sixth General Session

Little Theater, Worcester Memorial Auditorium

Dr. George L. Schadt, *Chairman*

Dr. John F. Casey, *Co-chairman*

2:00 *Current Trends in the Care of Depleted Surgical Patients.* Dr. Francis D. Moore, Boston. Moseley Professor of Surgery, Harvard Medical School; surgeon-in-chief, Peter Bent Brigham Hospital.

2:25 *Pitfalls in Anesthesiology.* Dr. Robert D. Dripps, Philadelphia. Professor of anesthesiology, University of Pennsylvania School of Medicine.

2:50 *Indications for Total Hysterectomy.* Dr. Bayard Carter, Durham, North Carolina. Professor of obstetrics and gynecology, Duke University School of Medicine.

3:15 *Obstetric Problems in Rural Districts.* Dr. Penry L. B. Ebbett, Houlton, Maine. Chief of staff, Aroostook General Hospital.

3:40 *The Management of Posterior Tuberculous Sinuses Permitting Early Spinal Fusion in Potts Disease.* Dr. Edward W. Boone, Rutland. Surgical Service, Veterans Hospital, Rutland. Drs. John J. Cincotti, chief of surgical service; Arnold M. Selzberg; John B. Kelley, consultant in orthopedics, Veterans Hospital, Rutland.

4:05 *The Use of Psychotherapy in General Practice.* Dr. Leo Alexander, Newton. Instructor in psychiatry, Tufts College Medical School; director, Neurobiologic Unit, Division of Psychiatric Research, Boston State Hospital.

4:30 *A Motion Picture Entitled "Cervicitis: Etiology, diagnosis and treatment."*

SCIENTIFIC EXHIBITS

BOOTH

79 — The Use of Tantalum Gauze in Various Forms of Hernioplasty.
Sponsor: Memorial Hospital, Worcester.
Exhibitor: Dr. George R. Dunlop.

80 — The Use of Cotton in the Open Technic Intestinal Anastomosis.
Sponsor: Memorial Hospital, Worcester.
Exhibitor: Dr. Donald Hight.

81 — Pantopaque Myelography in the Diagnosis of Protruded Intervertebral Disks.
Sponsor: Memorial Hospital, Worcester.
Exhibitors: Dr. William J. Elliott, Dr. James R. Lingley and Dr. John F. Sheehan.

82 — Chronic Pneumonitis — Cholesterol Type.
Sponsors: Massachusetts General Hospital, Boston, Memorial Hospital, Worcester.
Exhibitors: Dr. Laurence L. Robbins and Dr. Ronald C. Sniffen.

83–84 — Toxic Hazards in the Plastics Manufacturing Industry.
Sponsor: Plastics Division, Monsanto Chemical Company, Springfield
Exhibitor: Dr. Lamson Blaney.

85 — General and Plastic Surgery of the Eye.
Sponsor: Fairlawn Hospital, Worcester.
Exhibitor: Dr. E. Porter Jewett, Jr.

86 — Adenomyosis Uteri.
Sponsor: Fairlawn Hospital, Worcester.
Exhibitor: Dr. Arthur C. Brassau.

87 — Industrial Medicine, Worcester, Massachusetts.
Sponsors: Industrial physicians of Worcester.
Exhibitors: Dr. Karl T. Benedict and co-operating industrial physicians.

88 — Orthopedic Surgery in Children.
Sponsor: Worcester City Hospital.
Exhibitor: Dr. John W. O'Meara.

89 — Diseases of the Eye.
Sponsor: Worcester City Hospital.
Exhibitor: Dr. Elton Yasuna.

90 — General Surgery.
Sponsor: General Surgical Staff, Worcester City Hospital.
Exhibitors: Dr. Benjamin Andrews and staff.

95 — Endometriosis.
Sponsor: The Fallon Clinic, Worcester.
Exhibitors: Dr. John J. Manning, Dr. William G. Moran, and Dr. Elizabeth Fletcher.

96 — Diagnostic Discolorations of the Abdominal Wall.
Sponsor: The Fallon Clinic, Worcester.
Exhibitors: Dr. James T. Brosnan, Dr. John Meyers, and Dr. John Fallon.

97 — Fevers Uncommon in General Practice.
Sponsor: St. Vincent Hospital, Worcester.
Exhibitor: Medical Section.

98 — *Sponsor*: The Worcester Foundation for Experimental Biology.
Exhibitor: The Worcester Foundation for Experimental Biology.

99 — Hospital Rehabilitation.
Sponsor: Cushing Veterans Administration Hospital, Framingham.
Exhibitors: Physical Medicine Rehabilitation Service, Dr. Fritz Friedland, Chief, Paul D. Faden, Executive Assistant.

100 — Research in Epilepsy.
Sponsor: National Veterans Epilepsy Center, Cushing Veterans Administration Hospital, Framingham.
Exhibitors: Dr. Jerome K. Merlis, Dr. G. Kenriksen, and Dr. C. Grossman.

101 — The Program of Rehabilitation of the Tuberculous.
Sponsor: The Rutland Training Center.
Exhibitors: Dr. Oscar Feinsilver and Leonard F. Janes, M.Ed.

102 — Streptomycin in the Surgery of Tuberculosis.
Sponsor: Veterans Administration Hospital, Rutland Heights.
Exhibitors: Dr. John J. Cincotti and Dr. Edward W. Boone.

103 — The Treatment of Pulmonary Tuberculosis with Streptomycin.
Sponsor: Veterans Administration Hospital, Rutland Heights.
Exhibitors: Dr. Stanton T. Allison and Dr. Ralph Voll.

104-106 — Use of Transmetatarsal Amputation in Diabetic Gangrene.
Sponsor: Worcester Hahnemann Hospital.
Exhibitor: Dr. Elwood O. Horne.

1. Hip Nailing.
2. Modified Hibbs' Lumbo-Sacral Fusion.
3. Splints for Keller Bunionectomy.

Sponsor: Worcester Hahnemann Hospital.

Exhibitors: Dr. Ralph S. Perkins, Dr. Herman L. Matern, and Dr. Robert L. Moore, Jr.

Essentials of a Modern Nursery.
Sponsor: Worcester Hahnemann Hospital.
Exhibitor: Dr. Joel M. Melick.

Retropubic Prostatectomy.
Sponsor: Worcester Hahnemann Hospital.
Exhibitors: Dr. Lester M. Felton and Dr. Harold M. Constantian.

Modified Spinal Anesthesia in Cesarean Section.
Sponsor: Worcester Hahnemann Hospital.
Exhibitors: Dr. Anthony D. Vamvas and Dr. Constance B. Kaliris.

Corner 1

Physiology of Surgical Anesthesia.
Sponsor: Department of Anesthesia, St. Vincent Hospital.
Exhibitors: Dr. James C. McCann and Dr. William E. Martin, Jr.

Corner 2

Massachusetts Blue Shield.
Dr. Charles G. Hayden.

Corridor

Cancer of the Thyroid.
Sponsor: The Lahey Clinic, Boston.
Exhibitors: Dr. Frank H. Lahey, Dr. Hugh F. Hare and Dr. William A. Neissner.

Corridor

Springfield Municipal Hospital.
Sponsor: The City of Springfield.

Stage

An X-Ray Film Projector.
Exhibitors: Dr. Raymond A. Dillon, Doris D. Dillon, and Dr. William P. Murphy, Jr.

TECHNICAL EXHIBITORS

1949

	BOOTH NO.
Abbott Laboratories	78
Alkalol Company	55
Ames Company, Inc.	16
Atlantic X-Ray Company	70
Ayerst, McKenna & Harrison, Ltd.	2
Baker Laboratories	29
Beech-Nut Packing Company, Inc.	37
The Best Foods, Inc.	53
Bilhuber-Knoll Corporation	59
The Borden Company	15
Brewer & Company, Inc.	66 & 67
Brown & Connolly, Inc.	72
Buffington's Inc.	20
Burroughs Wellcome & Company, Inc.	33
Cambridge Instrument Company	45
Camel Cigarettes	17 & 18
Carnation Company	74
Ciba Pharmaceutical Products, Inc.	32
Coca-Cola Bottling Company of Worcester	93 & 94
Coreco Research Corporation	4
Crosbie-Macdonald	3
Davies, Rose & Company, Ltd.	7
Denver Chemical Manufacturing Company, Inc.	51
The Doho Chemical Corporation	75
Dy-Dee Service, Inc.	50
Edin Electronics Company	28
Endo Products, Inc.	11
C. B. Fleet Company	77
General Electric X-Ray Corporation	68
Gerber Products Company	24
J. E. Hanger, Inc.	31
Hanovia Chemical & Manufacturing Company	49
Harper X-Ray Company	13
Harrower Laboratory, Inc.	61
H. J. Heinz Company	43
Hoffmann-LaRoche, Inc.	30
The Junket Brand Foods	46
Kelley-Koett Manufacturing Company	62 & 63
George Laben	26
Lederle Laboratories Division	5
Eli Lilly and Company	1
M & R Dietetic Laboratories, Inc.	40
E. F. Mahady Company	8, 9 & 10
McIntosh Electric Corporation	76
McNeil Laboratories, Inc.	19
Mead Johnson & Company	36
Medical Clearing Bureau, Inc.	57
Medical Protective Company	12
National Drug Company	35
Nestlé Company, Inc.	34
Parke, Davis & Company	38
Pet Milk Sales Corporation	64 & 65
Philip Morris & Company, Ltd., Inc.	73
Picker X-Ray Corporation	47 & 48
Rare Chemicals, Inc.	60
L. & B. Reiner	22 & 23
Sandoz Chemical Works, Inc.	21
Saratoga Springs Authority	69
Schenley Laboratories, Inc.	14
Schering Corporation	71
G. D. Searle & Company	6
Sharp & Dohme, Inc.	42
Smith, Kline and French Laboratories	54
Spencer, Inc.	25
E. R. Squibb & Sons	56
Surgeons and Physicians Supply Company	91 & 92
Tailby-Nason Company	27
U. S. Vitamin Corporation	52
Vaponefrin Company	39
Varick Pharmacal Company, Inc.	44
White Laboratories, Inc.	58
Winthrop-Stearns, Inc.	41

MASSACHUSETTS MEDICO-LEGAL SOCIETY
ANNUAL MEETING

WEDNESDAY AFTERNOON, MAY 25

ROOM ONE, WORCESTER MEMORIAL AUDITORIUM

2:30 p.m.

PROGRAM

BUSINESS MEETING: Election of officers.

WAR CRIMES AND THEIR MOTIVATION. DR. LEO ALEXANDER, formerly special consultant to the Secretary of War.

Refreshments.

EXHIBITION OF WORKS OF ART
by members of the
MASSACHUSETTS PHYSICIANS' ART ASSOCIATION

ROOMS TWO AND THREE, WORCESTER MEMORIAL AUDITORIUM

Daily: 9:00 a.m.–5:00 p.m.

May 24, 25 and 26

ANNUAL GOLF TOURNAMENT

WORCESTER COUNTRY CLUB

WEDNESDAY AFTERNOON, MAY 25

1:00 p.m.

DR. FRANKLYN P. BOUSQUET, *Chairman*
390 Main Street
Worcester, Massachusetts

Luncheon may be obtained at the club

WOMAN'S AUXILIARY OF THE
MASSACHUSETTS MEDICAL SOCIETY

The registration desk will be located on the stage of the Worcester Memorial Auditorium. Registration will be from 9:00 a.m. to 5:00 p.m. on both days of the meeting.

TUESDAY MORNING, MAY 24

11:00 **Annual Meeting.**

The meeting will be held in Dean Hall, Worcester Woman's Club, entrance on Salisbury Street. (Buffet luncheon, at a charge of 95 cents, served at the club.)

TUESDAY AFTERNOON, MAY 24

2:00 **Visit to Worcester Historical Society, Art Museum and American Antiquarian Society.**

4:30 **Tea at Worcester Medical Library** (57 Cedar Street).

WEDNESDAY MORNING, MAY 25

10:30 **Bus Tour Starting from Worcester Memorial Auditorium.** (Luncheon at the Publick House; charge for bus tour and luncheon, $2.50.)

NOTICES

NEW ENGLAND CENTER HOSPITAL
(JOSEPH H. PRATT DIAGNOSTIC HOSPITAL)

30 Bennet Street, Boston
Lecture Hall, 9–10 a.m.

MEDICAL CONFERENCE PROGRAM

Friday, May 6 — The Importance of Hypothalamus to Adrenal Cortical Function. Dr. David M. Hume.

Wednesday, May 11 — Pediatric Clinicopathological Conference. Drs. James M. Baty and H. E. MacMahon.

Friday, May 13 — Experiences with Grafting Procedures in Vascular Surgery. Dr. Charles A. Hufnagel.

Tuesday, May 17 — Rheumatoid Spondylitis. Dr. Edward H. Fischer.

Friday, May 20 — Clinical Uses of Aureomycin. Dr. Harvey S. Collins.

Tuesday, May 24 — Recent Investigations in Virus Diseases. Dr. Lewis W. Kane.

Friday, May 27 — The Effect of Trimethadione (Tridione) and Other Drugs on the Major Convulsive Seizures Produced by Di-Isopropyl-Fluorophosphate (DFP). Dr. Harold E. Himwich.

Tuesday, May 31 — Biological Antagonists. Dr. John J. Finn.

From 9 to 10 a.m. on Wednesday (except the second Wednesday), Thursday and Saturday mornings, clinics will be given by members of the hospital staff. Medical Rounds are conducted each weekday except Saturday by members of the hospital staff from 12 to 1 p.m. On the second and fourth Fridays of the month, May 13 and 27, Therapeutic Conferences will be held with round-table discussion from 2 to 4 p.m. Dr. Robert P. McCombs as moderator. On the second and fourth Fridays, May 13 and 27, Dr. Merrill Sosman will conduct X-Ray Conferences from 4 to 6 p.m.

On Saturday mornings from 9 to 10 a.m. Surgical Clinics are conducted by Dr. C. Stuart Welch.

All exercises are open to the medical profession.

BOSTON CITY HOSPITAL
HOUSE OFFICERS' ASSOCIATION

The following program of the Boston City Hospital House Officers' Association will be presented in the Evening Lecture Series:

May 10. Precancerous Skin Lesions. Stephen Rothman, M.D., professor of dermatology, University of Chicago, the School of Medicine.

This program will be held at 7:00 p.m. in the New Cheever Amphitheater of the Dowling Building, Boston City Hospital.

All interested persons are invited to attend.

SOCIETY MEETINGS AND CONFERENCES

MAY 6-31. New England Center Hospital (Joseph H. Pratt Diagnostic Hospital). Medical Conference Program. Notice above.

MAY 7. New Engalnd Society of Anesthesiologists. Page 401, issue of March 10.

MAY 9. New England Heart Association. Page 668, issue of April 21.

MAY 10. Harvard Medical Society. Lower Out-Patient Department Amphitheater, Massachusetts General Hospital.

MAY 10. Harvard Medical Society. Page 702, issue of April 28.

MAY 10. New England Society of Anesthesiologists. Page 702, issue of April 28.

MAY 10. Boston City Hospital House Officers' Association. Notice above.

MAY 11. Harvard School of Public Health. Page 702, issue of April 28.

MAY 11. South Boston Medical Society. Page 702, issue of April 28.

MAY 11. Massachusetts Society of Examining Physicians. Page 702, issue of April 28.

MAY 11. Norfolk District Medical Society. Page 702, issue of April 28.

MAY 12. Chemotherapy of Leukemia and Lymphosarcoma. Dr. William Dameshek. Pentucket Association of Physicians. 8:30 p.m. Haverhill.

MAY 16-19. American Urological Association. Biltmore Hotel, Los Angeles, California.

MAY 18-21. Association for Physical and Mental Rehabilitation. Page 401, issue of March 10.

MAY 18-21. American Orthopaedic Association. Page 702, issue of April 28.

(*Notices concluded on page xix*)

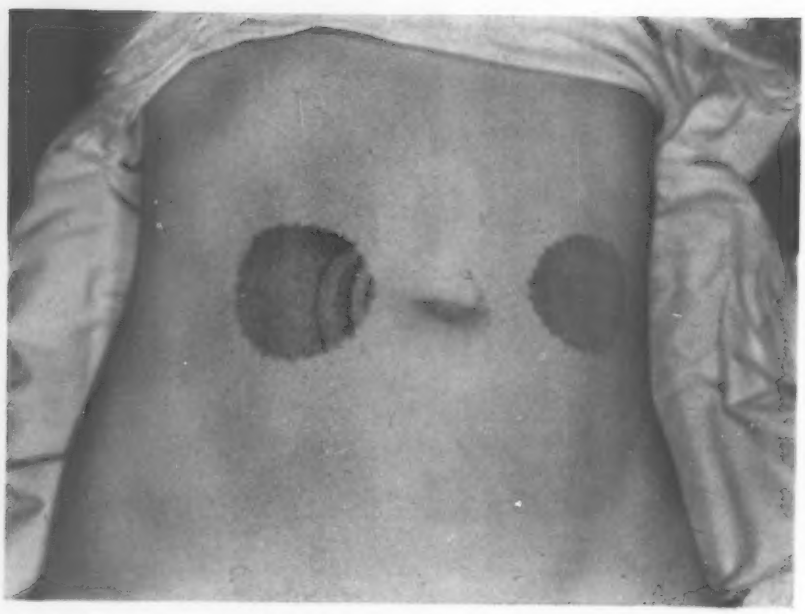

FIGURE 1. *A Variant of Cullen's Sign, Consisting of Two Paraumbilical Ecchymoses, Symmetrically Located over Points at Which Vessels Often Perforate the Anterior Rectus Sheath, and Another, Fainter Ecchymosis in the Umbilicus Itself.*

The photograph was taken eight hours after a sudden hemorrhage from ruptured tubal pregnancy.